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AND ABSTRACT OF THE LITERATURE

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Acid Burns and Their Treatment

Some Industrial Eye Affections

Health in Mercantile Establishments. II. Medical Records

A Statistical Study of Accidents in the Cotton Mills, Print Works and Worsted Mills of a Textile Company

Industrial Gassing and the Edward Medal

Experimental Trinitrotoluene Poisoning

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THE JOURNAL OF INDUSTRIAL HYGIENE

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ACID BURNS AND THEIR TREATMENT*

G. A. WELSH, M.D.

Medical Officer to H. M. Factory, Gretna

STATISTICS

AN analysis of the records at the dressing station on the acid section during the period from August 14, 1916, to September 30, 1919, shows that one out of every four cases was treated for an acid burn; the actual number of cases treated at the station amounts to 17,414; acid burns account for 4,292. A more detailed analysis of the acid burns showing the plant of incidence and classifying the degree of the burn gives the following figures:

	Slight	Severe	Serious
Grillo oleum	154	16	5
Mannheim oleum....	62	12	4
Nitric acid retorts...	1117	42	10
Nitric acid mixers ...	294	24	9
Nitric acid stills.....	410	18	3
De-nitrators	116	4	2
Gaillard towers.....	170	14	..
Laboratories	84	9	2
Nitro cotton nitrating	1621	87	3
	4028	226	38

The definition of the terms used is as follows:

Slight. — A burn which does not incapacitate the worker for work or which is healed and allows the worker to return to work under ten days.

Severe. — A burn which incapacitates the worker for fourteen days or over, and which varies in degree up to one which incapacitates the worker for as much as four weeks.

Serious. — A burn by which the worker is incapacitated for over four weeks, and which in a large proportion of cases leaves a degree of permanent incapacity.

To illustrate this I append an analysis of the compensation list record showing the occupation, plant of incidence, nature of the acid causing the injury, part affected and period of incapacity (Tables 1 and 2) in ninety cases of acid burns.

GENERAL CONSIDERATIONS

A condition which causes 25 per cent. of the cases dealt with arrests the attention, quite apart from the intrinsic importance of the subject. One outstanding feature of these figures is the large proportion of cases which are classified as slight. This speaks very much in favor of the thoroughness of the precautions taken to prevent accident and shows that an efficient first-aid system is of real value.

* Received for publication June 21, 1920.

In dealing with the subject of acid burns in a factory such as H. M. Factory, Gretna, where the production of acid is on a large scale and constitutes an engineering as well as a chemical problem, I propose to do so by introducing short descriptions of things not purely surgical, which will elucidate the points I want to emphasize. For the purpose of description I have divided those working the plant into: (a) operatives whose work is chiefly chemical, and (b) operatives whose work is chiefly mechanical. In practice it is, however, difficult to maintain such a hard and fast distinction. In the case of operatives whose work is chiefly chemical, it is possible to protect the exposed surfaces — e. g., the hands by rubber gloves and the eyes by goggles. In the case of operatives whose work is chiefly mechanical, this is not possible as the workman is using tools and cannot work so efficiently if his touch is dulled or his sight in any way blurred. This was the attitude of the workmen and made it very difficult to adopt precautionary measures. The fact is important because in the majority of cases the escape of acid from a closed system was due to an accident closely connected with a breakdown in the machinery. It is also germane to mention that the escape of acid was often sudden and with considerable force behind it, due to the pressure in the system.

In the process of cotton nitration we have an illustration of how the mechanical element enters into the work of a chemical operative. We have also an example of the value of protecting exposed surfaces. To prepare the pan for nitrating, the operative has to push the cotton into the mixed acid with a long handled fork, and, when the pan is full, to place in position the earthenware top. Both of these operations are mechanical and if not carefully performed displace the acid and cause splashing. In 96 per cent. of the cases from the nitrating house, the acid burn was caused by the acid splashing.

The mechanical operative in the ordinary course of employment soils his hands and when an accident happens the acid comes in contact with the dirty surface — in many instances with abrasions and cuts. It did not take me many visits of inspection to see how dirty the hands of some of the workmen were and to appreciate how difficult it would be to prevent sepsis. The importance of this point was speedily impressed upon me; in the early days I saw numerous cases where the acid burn was so slight it gave the workman no concern at the time. He only reported for treatment when sepsis developed and this caused an unnecessary prolongation of the period of incapacity. To remedy this, I recommended the section manager to issue instructions for every case of acid burn to report at the dressing station. The nurse had instructions to clean the burned part and surroundings thoroughly before applying the final dressing. It took some time for this advice to be appreciated at its proper value but eventually there was a general conversion and many hours of labor were saved to the factory, and a smaller compensation list resulted. Summarizing, I wish to emphasize three points: (1) that it is of prime importance to protect as far as possible exposed surfaces — e. g., the hands and the eyes; (2) that it is essential to take every step to avoid sepsis; (3) that the operatives should be taught to look after the state of their hands and pay special attention to cuts and abrasions.

Varieties of Acid

The acids manufactured are: concentrated sulphuric acid, oleum, and concentrated nitric acid. These acids are mixed in varying proportions to produce mixed acids for nitrating purposes. In addition to these, the spent acids left after nitrating are de-nitrated and weak sulphuric acid and nitric acid recovered for further use.

TABLE 1. — LIST OF INJURIES CAUSED TO MALE WORKERS ON THE ACID SECTION BY ACID BURNS

Case	Occupation	Plant where injury happened	Nature of acid causing injury	Part affected by burn	Period of incapacity in weeks
1	fitter's helper	nitric acid retorts	mixed acid	face	1
2	chemical operative	nitric acid retorts	cold nitric	eye	1
3	chemical operative	Gaillard towers	cold weak sulphuric	both eyes: r. slight; l. mod. severe. Face and forehead slight	1
4	chemical operative	acid mixers	concentrated sulphuric	acid splash in eye	1
5	chemical operative	acid mixers	weak cold sulphuric	face	1
6	leadburner	nitric acid retorts	cold strong nitric	face	1
7	chemical operative	nitric acid stills	cold nitric	right eye	1
8	chemical operative	Mannheim oleum	cold weak sulphuric	face	2
9	chemical operative	nitro cotton mixers	cold nitro cotton mixed	face and eyelids	2
10	laborer	Gaillard towers	cold weak sulphuric	hand	2
11	chemical operative	acid mixers	cold oleum	face	2
12	fitter's helper	grillo oleum plant	cold sulphuric	arm	2
13	chemical operative	nitro cotton mixers	cold nitro cotton mixed	wrist and thigh	2
14	laboratory chemist	laboratory	cold strong sulphuric	eye	2
15	chemical operative	acid mixing	cold oleum	neck	3
16	chemist's assistant	sulphuric acid laboratory	concentrated cold sulphuric	arm	3
17	laborer	oleum weigh house	cold strong sulphuric	side of neck and ear	3
18	gas producer operative	nitric acid stills	weak sulphuric	foot	3
19	fitter	acid mixers	cold mixed acid	finger	3
20	chemical operative	nitric acid stills	cold nitric	face	3
21	fitter's helper	acid mixers	cold mixed acid	eye	3
22	chemical operative	acid mixers	cold nitroglycerine mixed	face	3
23	chemical operative	nitric acid retorts	molten nitrecake	hand	3
24	chemical operative	nitric acid retorts	molten nitrecake	hand	3
25	chemical operative	nitric acid retorts	molten nitrecake	right eye	3
26	chemical operative	nitric acid retorts	cold nitric	finger	3
27	engineer	nitric acid stills	hot sulphuric	leg	3
28	chemical operative	grillo oleum	cold oleum	face and neck, l. eyelid and eye	4
29	chemical operative	Mannheim oleum	cold oleum	arm	4
30	fitter	grillo oleum	cold oleum	face and neck	4
31	leadburner	de-nitrators	spent nitroglycerine	head and neck	4
32	chemical operative	nitric acid retorts	cold strong nitric	left arm	4
33	chemical operative	nitric acid retorts	cold strong nitric	foot	4
34	chemical operative	nitric acid retorts	hot nitrecake	hand	4
35	bricklayer	nitric acid stills	cold strong nitric	hand	4
36	chemical operative	nitric acid retorts	hot nitrecake	hand and arm	4
37	chemical operative	grillo	sulphuric	hand	5
38	chemical operative	nitric acid retorts	molten nitrecake	arm	5
39	chemical operative	nitric acid retorts	molten nitrecake	hand and arm	6
40	chemical operative	acid mixing	weak nitric	foot; burn neglected	7
41	chemical operative	nitric acid retorts	cold strong nitric	hand	7½
42	fitter	grillo oleum	cold strong sulphuric	face, neck and arm	8
43	chemical operative	acid mixing	cold oleum	leg	8
44	fitter	oleum weigh house	cold strong sulphuric	face, eyes, forehead and l. arm	9
45	chemical operative	nitric acid stills	cold strong nitric	1st finger l. hand: sepsis followed causing necrosis and loss of 1st phalanx	10
46	chemical operative	nitric acid retorts	hot strong nitric	foot	15
47	chemical operative	acid mixing	cold mixed nitroglycerine	both hands, lower part of back and both buttocks	17
48	chemical operative	acid mixing	cold mixed nitroglycerine	face, arms and back. Severe burns affecting both eyes; l. eye became septic and had to be excised	30
49	chemical operative	acid mixing	cold strong nitric	severe burn of l. eye, head and face and eventually loss of sight of l. eye; incapacitated long period. Claim settled by payment of £300	
50	chemical operative	acid mixers	cold strong sulphuric	eyes and face; l. eye, which had been seat of old injury, became septic and man died of septic meningitis	
51	chemical operative	Mannheim oleum	cold strong sulphuric	acid burn affecting eyes, eyelids and face; the operative is totally incapacitated and has developed tuberculosis	

TABLE 2. — LIST OF INJURIES CAUSED TO FEMALE WORKERS ON THE ACID SECTION BY ACID BURNS

Case	Occupation	Plant where injury happened	Nature of acid causing injury	Part affected by burn	Period of incapacity in weeks
1	chemical operative	nitrating house	nitro cotton mixed acid	right hand	1
2	chemical operative	nitrating house	nitro cotton mixed acid	acid splash, both eyes	1
3	chemical operative	nitrating house	nitro cotton mixed acid	arm	1
4	chemical operative	nitrating house	nitro cotton mixed acid	foot	1
5	chemical operative	grillo oleum	strong cold sulphuric	shoulders, neck and face; acid splash in eye	2
6	chemical operative	nitrating house	nitro cotton mixed acid	left forearm	2
7	chemical operative	nitrating house	nitro cotton mixed acid	eye	2
8	chemical operative	nitrating house	nitro cotton mixed acid	both feet	2
9	chemical operative	nitrating house	nitro cotton mixed acid	left thigh and ankle	2
10	chemical operative	nitrating house	nitro cotton mixed acid	arm	2
11	chemical operative	nitrating house	nitro cotton mixed acid	foot	2
12	chemical operative	nitrating house	nitro cotton mixed acid	foot	2
13	chemical operative	nitrating house	nitro cotton mixed acid	eyes	2
14	chemical operative	nitrating house	nitro cotton mixed acid	fingers	2
15	chemical operative	nitrating house	nitro cotton mixed acid	hands	2
16	chemical operative	nitrating house	nitro cotton mixed acid	both eyes	2
17	chemical operative	nitrating house	nitro cotton mixed acid	hands and knees	3
18	chemical operative	nitrating house	nitro cotton mixed acid	right arm	3
19	chemical operative	nitrating house	nitro cotton mixed acid	finger	3
20	chemical operative	nitrating house	nitro cotton mixed acid	arm	3
21	chemical operative	nitrating house	nitro cotton mixed acid	wrist	3
22	chemical operative	nitrating house	nitro cotton mixed acid	fingers and eye	3
23	chemical operative	grillo oleum	cold oleum	eye	4
24	chemical operative	nitrating house	nitro cotton mixed acid	left instep	4
25	chemical operative	nitrating house	nitro cotton mixed acid	wrist	4
26	chemical operative	nitrating house	nitro cotton mixed acid	leg	4
27	chemical operative	nitrating house	nitro cotton mixed acid	hands	4
28	chemical operative	nitrating house	nitro cotton mixed acid	foot	5
29	chemical operative	nitrating house	nitro cotton mixed acid	arm	5
30	chemical operative	nitrating house	nitro cotton mixed acid	foot	5
31	chemical operative	nitrating house	nitro cotton mixed acid	back	5
32	chemical operative	nitrating house	nitro cotton mixed acid	foot	6
33	chemical operative	nitrating house	nitro cotton mixed acid	foot	6
34	chemical operative	nitrating house	nitro cotton mixed acid	r. instep, septic	7 w. 4 d.
35	chemical operative	nitrating house	nitro cotton mixed acid	burn of arm	12
36	chemical operative	nitrating house	nitro cotton mixed acid	eye; sight of eye injured; since died from pneumonia	15
37	chemical operative	nitrating house	nitro cotton mixed acid	acid burn injuring sight of r. eye. Settled by payment of £100	over a year
38	chemical operative	nitrating house	nitro cotton mixed acid	burn of eye causing loss of sight. Settled by payment of £100	over a year
39	chemical operative	nitrating house	nitro cotton mixed acid	cut finger from cracked nitrating pan, burned at same time with nitro cotton acid	24

During the course of this de-nitration process, the temperature of the acid is raised to a considerable degree. In the case of weak sulphuric acid a temperature of 200°C. is reached. Lastly there is a by-product obtained from the manufacture of nitric acid, an acid sulphate of soda called nitrecake, which is discharged from the retorts in a molten state at about 150°C. to cooling pans. It follows that an operative may be burned with oleum alone, with sulphuric acid alone, with nitric acid alone, with mixed acid, or with nitrecake at a high temperature.

External Appearances of Acid Burns

It is possible to recognise by external appearances the action of these different acids on the skin. Oleum and sulphuric

acid produce a grayish-brown color; nitric acid produces a bright yellow discoloration; mixed acid produces a reddish-brown color with a faint yellow in it, the yellow being most distinguishable at the margin of the burn. There is, in the early stages, little congestion of the surface when there is no heat acting with the acid. When a burn is inflicted with an acid at a high temperature, as with the molten nitrecake, there is well-marked congestion and bullae are quickly formed. This congestion masks the colors produced by the same acid in the cold state. For purposes of treatment it makes no difference what cold acid produces the burn, and there is only a difference in technique, not in principle, when the part is scalded as well as burned by acid. Concentrated sulphuric acid or concentrated nitric acid falling on the skin of the hands and speedily treated will produce very little effect; a weaker acid with heat produces a burn which gives more trouble. Burns of a mucous surface — e. g., burns of the lips and inside of the mouth, and of the eye — cause a marked reaction and take longer to heal.

Treatment

In dealing with an acid burn everything depends upon the promptness and thoroughness of the first steps taken to treat it. We have here a first-rate example of the importance of what is known as first aid. To ensure promptness of treatment in H. M. Factory at Gretna, a brief description of what should be done was printed and circulated to the section managers and those in authority acting under them. Eventually these rules were incorporated in the factory rule book so that each operative might have the information. The instructions issued were as follows:

> Get rid of surface acid by the application of clean water to the part; this may be done by immersing the part in a tub of water, by playing a hose on it, or by leaving water on it if it is the face or head. Neutralize the acid in the part by applying the alkaline solution and allowing it to act for ten minutes. Gently dry with cotton wool and apply moist picric acid gauze. Cover the surface with a layer of cotton wool and keep in position with a bandage lightly applied. The person who is burned should not be allowed to move the affected part. If the arm is burned a sling should be applied; if the legs are burned a stretcher should be used. The patient should be seen by the doctor as soon as possible. When the part affected is the eye or the mouth, it should be washed with alkaline solution and oil applied over the surface.

First-aid boxes containing the necessary articles for treatment and buckets of clean water were placed all over the plant within easy reach of every operative. These appliances were regularly inspected and an ample supply maintained. In addition to this, as I mentioned earlier, every case was reported to the nurse, who inspected simple cases and reported all severe cases to the doctor. All severe cases had the final dressing applied by the doctor. Cases where a mucous surface was affected or the eye burned were personally treated by the doctor.

The dressing station is placed within three minutes' walk of the most outlying part of the acid section, which is the grillo plant. All the other plants are within half that distance. This station is well heated, is equipped with sterilizing apparatus, has ample bath accommodation and four beds for the detention of any serious cases.

No patient is brought to the dressing station until all surface acid has been got rid of by ample treatment with cold water. In the case of the plant adjoining the station, further stages of treatment are carried out at the dressing station: in the case of an outlying plant, the dressing is applied and removed on arrival for inspection of the burn.

In the cases here reported the nurse obtained the history on arrival of the patient, and if the burn was a simple one dressed it

and reported afterwards. In severe cases the nurse reported by telephone and proceeded to prepare the patient for examination, being particularly careful to remove all acid-soaked clothing before cleaning up the affected area and surroundings. Before dealing further locally with a severe case — e.g., a case in which an extensive surface was affected, or, a special area (the head, face and neck, both eyes or one eye) badly affected — I always took precautionary measures to combat shock. The patient was treated in a temperature between 65° and 70°F.; he was given a hot drink, milk or bovril, and a sixtieth of a grain of strychnine hypodermically. The cleaning and soaking of the burned area with alkaline solution having been thoroughly attended to, moist picric acid gauze (strength 1 per cent.) was applied and the part covered with sterilized cotton wool and lightly bandaged. The alkaline solution which I used was a saturated solution of recrystallized sodium carbonate. This I found to be a very useful agent for cleaning the part as well as neutralizing any free acid in the tissue. During the acute stage of the burn the dressing was changed twice daily, moist picric acid gauze being re-applied over the area. This was continued until healing was well advanced. This dressing was then changed for sterile plain gauze wrung out of normal saline solution. If the covering of the surface with epidermis was in any way delayed, I applied sterile gauze soaked in ten volumes of solution of hydrogen peroxide. In cases where sepsis developed I substituted for the picric acid gauze, gauze soaked in hydrogen peroxide, the pus being gently swabbed away from the surface with normal saline solution. When blisters were present over the burned area I left them untouched for twenty-four hours; they were then pricked, care being taken to make a small opening, and as much of the fluid as could be removed was dried off. On or about the third day, the superficial layer was excised by means of scissors and the picric acid dressing continued.

Eye Cases

External Appearances. — In the case of a moderately severe eye burn, the external appearances consist in edema of the conjunctiva with well-marked injection of the burned part and its surroundings. Severe pain is complained of and photophobia is marked. If a concentrated acid gains entrance to the eye in any quantity there is very quickly developed stasis in the corneal lymphatic system and the cornea becomes quite milky, even to the extent of the obliteration of the pupil.

Treatment. — As soon as possible after the burn was received in the cases reported, the whole of the conjunctival sac was douched with the saturated solution of sodium carbonate. In the majority of cases this was done in the plant. The further treatment consisted in instilling three or four drops of 1 per cent. solution of atropine sulphate and when the pupil became dilated a few drops of sterile castor oil. The eye was then covered with a pad soaked in boracic solution and a bandage applied. Further treatment consisted in washing out the eye night and morning with boric solution and once in twenty-four hours instilling a few drops of sterile castor oil into the sac.

Before leaving the treatment of acid burns it may be useful if I shortly summarize the points I consider important, and at the same time explain the general principles on which the treatment is based. An acid progressively destroys the tissue so long as it is potent and only ceases to act when its corrosive property is destroyed. The more quickly you get rid of surface acid and the more thoroughly you render the acid in the tissue inert by neutralizing it, the more you limit the ultimate damage done. The cicatrix left after healing is in

direct proportion to the amount of tissue destroyed, and I need scarcely add that in the case of the face and eyelids this is a matter of great importance. The thorough cleaning of the burned area and its surroundings are steps taken to prevent sepsis with its attendant danger of absorption and systemic poisoning, the production of weak tissue in healing and the formation of a dense and more unsightly cicatrix.

In the dressing of the wound the substance used should be antiseptic and as little irritating as possible; it should not be oily in character nor should any greasy substance be applied to the skin surface. Pain is better dealt with by local application if possible, and the use of narcotics should be restricted to very severe cases. Picric acid is an ideal substance; its action in precipitating and combining with the albumin of the tissue forms a covering which is antiseptic and protective and at the same time allays pain. It has a property like that of carbolic acid in finding its way into the deeper tissue and acting there. I prefer to use it as a gauze and not as a solution painted on the surface. Its action is really on the same principle as the French substance, ambrine, in sealing up the surface of the damaged area with an antiseptic layer. In the case of ambrine, it is essential to dry the surface. Otherwise, the temperature at which it is applied in the presence of moisture will scald the tissue. At the stage in the treatment of acid burns where the tissue has been soaked with an alkaline solution to neutralize the acid, it is not possible, nor is it desirable, to attempt to dry the surface, and for this reason I think a better result is obtained from picric acid, which can be applied at a safe temperature. The final results of the picric acid treatment are very satisfactory and it is rare to have to graft skin. I have not seen any cases where cheloid developed in the cicatrix. In several of the cases where eyelids were affected, a plastic operation to relieve conditions like ectropion has been necessary. In the treatment of eye cases, I find that atropine relieves pain quite as much as cocaine, and has the advantage that its dilatation of the pupil is more lasting.

SOME INDUSTRIAL EYE AFFECTIONS*

JOHN C. BRIDGE, F.R.C.S.E., D.P.H.
His Majesty's Medical Inspector of Factories

THE lighting of factories and workshops in Great Britain was considered by a departmental committee which, after hearing evidence and carrying out investigations, issued their report in 1915 and recommended standards for efficient lighting (1). These standards have not yet been adopted, but the importance of good lighting for this class of building is undoubtedly receiving greater attention, not only on account of the harmful effects bad lighting may have on the eyes of the workers, but also because of the improved work, greater output, and lessened fatigue which necessarily follow lighting in its best form. So far, however, progress is slow and is largely confined to new buildings; to those familiar with industry, examples of lighting, bad in quality, quantity, and direction, are still innumerable. Not uncommonly a worker is found with his back to the light, using a poor artificial light where, by some rearrangement of his work, sufficient and good light could be obtained from a window.

A recent case seen by the writer also illustrates how actual economy can be effected by considering the question of satisfactory lighting. A man was found working at a sand-blasting cabinet, illuminated by an electric light placed within it. The cabinet was situated beside a large window so that the light from it was directed across the worker's eyes, thus preventing him seeing his work. To obviate the glare from the window the workman had covered it with sacking, brown paper, etc., making the room dark and the use of electric light in the room necessary for general illumination. As the cabinet was firmly fixed, the remedy was to fix a shield or "blinker" on the side of the cabinet towards the window. The removal of the window obstruction not only let in light and air, but also economised the electric light which previously was burning all day.

When educationalists realised that educational establishments, while educating, need not cripple the student, attention was given to the importance of proper lighting of the classroom. The time is not far distant, we hope, when the same consideration will be shown to the eyes of the industrial worker.

It is impossible to estimate to what extent the absence of proper lighting has affected the eyes of workers by producing or increasing refractive errors. The economic benefits of good lighting are, however, so obvious that figures are hardly necessary — if they could be obtained.

Apart from errors of refraction, eye strain and fatigue produced by bad lighting in factories and workshops, there are other definite diseases of the eye caused by certain forms of employment, and the object of this article is to summarise briefly some of those of interest.

Conjunctiva

Hyperemia of the conjunctiva is very frequently seen in workers exposed to acid fumes. Those most affected are men employed in the manufacture of the mineral acids, particularly hydrochloric acid. Exposure to the fumes of acids in other branches of the chemical trade — for example, the manufacture of dyes and their intermediate products — also gives rise to this condition. The halogen group, chlorine, bromine and iodine, produce a similar

* Received for publication July 20, 1920.

hyperemia, but the less frequent use of them makes such cases less common. Ammonia if present in any quantity in the atmosphere similarly affects the conjunctiva.

Acute inflammation of the conjunctiva is less frequent but may be caused by any of the substances mentioned above if the exposure is long enough or the concentration of the gas in the air high. Men employed in patent manure works, digging out the dens or pits, suffer from conjunctivitis caused by hydrogen fluoride, and etchers on glass are affected by the hydrofluoric acid used in the process. Hydrogen sulphide is stated to have been one of the causes of inflammation of the conjunctiva in the manufacture of matches from sesquisulphide of phosphorus (2), and the same gas is probably, to a large extent, responsible for this condition observed among sewer men.

In the dyeing industry, where sulphur dioxide is used, a similar affection has been observed, and Rambousek states in regard to this gas that even in low concentrations in the atmosphere it produces inflammation of the eyes (3).

Naphtha used as a substitute for alcohol in wood polishes has been known to give rise to the same condition, though workers employed spreading cloth with rubber dissolved in naphtha for the purpose of waterproofing and using large quantities daily have not been observed to be affected. Smarting and lachrymation does occur, however, on first entering a workroom where this work is in progress. The fumes from methyl alcohol used in denaturing spirits give rise to the same symptoms and in severe cases to conjunctivitis. Numerous severe and acute cases of conjunctivitis occurred during the war among persons employed in the manufacture of shells and in filling shells with chemicals — the chief of which was di-chlor-ethyl-sulphide — the fumes from which were specifically intended for this purpose. This cause, it is hoped, may be now regarded only as of historical interest.

Acute conjunctivitis is also caused by the light from the arc formed in electric welding, such light having an excess of ultra-violet rays. In these cases the exposure need only be equivalent to a flash if received close enough. The symptoms, which are severe, are fortunately merely temporary and commence some six to eight hours after exposure, the sensation of sand in the eye being the earliest; the pain, photophobia and lachrymation which follow are intense and are generally accompanied by headache. Remedial measures on the usual lines — cold applications with protection from light — are generally sufficient to give relief; and for the subsequent inflammation a mild astringent lotion is useful. Tinted glasses consisting of a ruby between two blues are necessary in order to protect the eyes, while screens to cut off flashes from the sides are also required. Acetylene welding, though giving rise to a bright light from which it is necessary to protect the eyes of the workers by blue or smoked glasses in order to prevent fatigue, does not produce an inflammatory condition, and in a worker with normal vision no after-effects have been observed beyond "eye tiredness." It is obviously desirable that workers exposed to such lights should be submitted to a preliminary eye test in order to determine the presence of errors of refraction which would increase the strain, but no special test can be employed. In this connection, the increasing use of one-half-watt lamps in factories, the unshaded light from which produces hyperemia of the conjunctiva, calls for comment.

Dust from a large number of chemicals causes inflammation of the conjunctiva by reason of the irritative nature of the chemical, but fortunately conjunctivitis from this source is not common. The basic aniline dyes are said to be specially liable

to cause injury. Para-nitraniline, used largely for dyes and photographic developers, sets up a very acute inflammation, as does also the explosive tetryl (tetra-nitro-methyl-aniline). Trinitro-toluene on the other hand, though like tetryl in producing an acute dermatitis, rarely affects the eye. Pitch dust produces severe conjunctivitis, which persists for some days; patent fuel workers and men loading and unloading pitch are principally affected.

Conjunctivitis produced by vegetable dust is practically always caused by mechanical irritation, though the dust of certain woods, notably West African and East Indian boxwood, appears to contain an alkaloid which in itself produces inflammation of the conjunctiva accompanied by severe lachrymation. Certain alkaloids themselves, emetine in particular, cause conjunctivitis among those preparing them for medicinal use. Two cases have come under notice of a peculiar susceptibility to linseed meal in oil-cake mills. In these patients the conjunctiva rapidly became suffused, pain and lachrymation were intense in one case, and the symptoms persisted for some days accompanied by coryza. Fresh entry into the mill on recovery produced a similar train of symptoms.

Mercury and silver fulminate both set up inflammation of the conjunctiva, the former causing inflammation of a severe character. Several outbreaks of severe conjunctivitis occurred in a factory where percussion caps of fulminate of mercury were being manufactured. This type of conjunctivitis is accompanied by pain and profuse lachrymation. Strict attention should be paid to personal cleanliness and the workers should be supplied with separate towels; while bathing the hands in a solution of hyposulphite of soda — a very weak solution of which may also be used for the eyes — is also beneficial. In the manufacture of snaps for crackers with fulminate of silver, the inflammation is very much less, but permanent staining of the conjunctiva (argyria) has been observed. In one case seen the employment extended over one year only; in those employed longer the pigmentation was most noticeable, staining of the whole conjunctiva being very well marked, particularly towards the inner canthus. Much of this work is done in small domestic workshops in surroundings where precautionary measures are impossible to enforce, the workers themselves having no very high standard of personal cleanliness. Pigmentation of the skin was also observed. It is probable in these cases that the salt is conveyed to the eyes by contaminated fingers. Women employed in polishing silver articles on polishing wheels also suffer from conjunctival pigmentation, as do men on plants for the desilverising of lead.

Conjunctival hemorrhages have been observed in cases of lead poisoning (4).

It is only necessary to mention the inflammation set up by splashes of acids and caustics into the eye, and the need for immediate treatment in such cases.

Cornea

Certain basic aniline dyes are said to affect the cornea, and in particular the dust of methyl violet which produces inflammation (5). Though strictly speaking the exciting cause of corneal ulceration occurring among patent fuel workers is the injury to the cornea by the spicule of pitch, the extent of the ulceration and the hypopyon which commonly follows must be considered to be due in some measure to the same constituent of the pitch as produces cancer of the skin among these workers. Tar and paraffin workers may be similarly affected. Inflammation and opacity of the cornea are said to be caused by nitro-naphthalene, attributable either to long continued exposure to the vapor or to spurting of the liquid into the eye (6).

Burns from splashes of acids and caustics which produce inflammation and ulceration of the cornea occur wherever such chemicals are handled and call for immediate first-aid treatment.

Retina

The commonest industrial cause of disease of the retina is lead poisoning. Legge and Goadby regard temporary or sudden blindness as due to a vascular change of the retina, either vasomotor or hemorrhagic (7). In advanced cases they state the "picture is one of severe albuminuric retinitis." Dilatation of the vessels has been observed among electric welders accidentally exposed to the light of the arc. Temporary and partial amaurosis is also produced. Retinitis and other eye changes are also described as occurring among this class of workers (8).

Toxic amblyopia, which may be considered under this heading, occurs among workers exposed to dinitrobenzene, those most affected being employed in filling cartridges. A few cases of this nature have been observed among T.N.T. workers but in these it was difficult to eliminate tobacco. The fumes of carbon bisulphide used in vulcanising rubber by the "cold cure" process may also produce similar symptoms. In such cases of amblyopia, the scotoma is central and that for green larger than red, both eyes being equally affected. Recovery usually takes place quickly after removal from the poison. Folker has described five cases of women suffering from amblyopia due to lead. Additional interest in these cases, as he says, lies in the fact that the use of tobacco could be safely excluded (9).

Pupil

The pupil appears to be rarely affected. Among pharmaceutical workers, accidental contact of the hands and eyes during the preparation of atropine salts produces the usual mydriatic effect. Instances of intolerance of the skin to morphia in the manufacture of the salts of this alkaloid are not accompanied by alteration in the pupil. In lead poisoning inequality of the pupil may be observed but partial dilatation of both pupils is more common (10).

Optic Nerve

Neuritis of the optic nerve occurs with chronic lead poisoning. It is usually, if not always, associated with retinitis, and total blindness occasionally occurs. Chronic nitrobenzene poisoning is stated to give rise to optic neuritis (11).

Lens

Glass bottle blowers suffer from posterior cortical cataract. Such workers, it was estimated by Dr. W. Robinson of Sunderland, are exposed to the glare of the molten metal for $5\frac{1}{2}$ hours per week, and it is now generally accepted, after the work on the subject by Sir William Crookes, that the heat given off from the metal is mainly, if not entirely responsible (12).

Several cases of cataract have been observed following accidental electric shock. These are due to the passage of the current through the body and not to the flash which sometimes accompanies an accident of this character (13).

Ocular Muscles

Paralysis of the ocular muscles was noted by Lockhart Gibson in Queensland among children accidentally poisoned by lead.

Nystagmus

Oscillation of the eyeball among miners is a well-recognised condition, the exact causation being at present not fully understood, although imperfect illumination of the coal face is generally regarded as an important factor. Reference must also be made to nystagmus which sometimes occurs in cases of lead poisoning (14).

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HEALTH IN MERCANTILE ESTABLISHMENTS
II. MEDICAL RECORDS*

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THE necessity for records has been discussed in the first paper of this series, but the precise form and the manner of introducing a record system have not been approached.

From the practical point of view of the store administrator, the subject divides itself into two parts. First, there is the question of the physical examination for employment, and, second, the record to be kept of an ordinary visit to the doctor's office. To many stores the policy of introducing a physical examination for employment seems at present inadvisable. They fear difficulties with their employees which may far offset any advantages that such modern methods may supply. There are very few well-organized industries employing large numbers of people who do not first estimate by physical examination the fitness of the employee for the work to be undertaken. It would seem the logical and intelligent policy, therefore, that stores should supplement other employment methods by instituting a preliminary examination of sufficiently broad scope to insure fitness to carry on the varied tasks of the store under the active conditions of city life.

We are personally acquainted with the work of five large stores in the East and Middle West which have been using examinations before employment for periods of from one to eight years. None of these have ever given up the procedure and all have realized its advantages more and more. Investigation shows that employment managers in many other stores are looking forward to the time when they, too, may have assurance from a modern medical department that the applicant is reasonably suited for the job. At present, they must depend on the general appearance and the prejudiced statements of the hopeful applicant, or upon the guarded reference of a former employer.

The practical benefits which the stores using physical examinations report include the steady improvement of their personnel due to better adaptation of the individual to his work, the discovery of serious disabilities, such as heart disease, so that if the applicant is accepted he is safeguarded, the exclusion of early cases of tuberculosis, and the detection of remediable defects, such as hernia, diseases of the mouth and teeth, etc. While the medical examination must result in preventing the employment of a certain number of persons, this has become a minor feature of its work. If the physician has knowledge of the different types of work which the store furnishes, he will be able to suggest places fitting the physical qualifications of nearly all applicants.

Industry presents some interesting examples in this direction. Clark (1) speaking from his experience in the Norton Company (manufacturers of grinding wheels and grinding machinery in Worcester, Mass.), where the restrictive list of defects is rather severe because of the heavy nature of the work, finds that 3.5 per cent. of those applying for work are rejected on physical grounds. Other reports show as high a figure as 10 per cent., but it seems probable that with thorough knowledge of the work of the plant the industrial physician can reduce this very markedly.

Such benefits as have been listed reach

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FIGURE 1

PHYSICAL EXAMINATION

Date..... 192....., ...M.

Name..... Check No..... Department.....
Nature of Work.....
Address..... Age..... Sex.....
Married..... Children..... Other Dependents.....
General Appearance..... How long in our employ.....
Weight..... Height..... Nationality..... Color.....

HEAD AND NECK: Evidence of previous injuries.....
.....

EYES: Vision close.....
Vision Distance, Right.....Left.....
Pupils, Size .....Shape.....
Reaction Light .....
Accommodation .....
Use of Glasses.....

EARS: Hearing, Right.....Left .....
Discharge, Right.....Left .....
Tympanic Membrane, Right .....Left.....

NOSE: Septum .....
Adenoids .....

THROAT: Tonsils .....
Tongue .....
Mouth.....
Teeth, Right $\frac{8\,7\,6\,5\,4\,3\,2\,1}{8\,7\,6\,5\,4\,3\,2\,1}$ Left $\frac{1\,2\,3\,4\,5\,6\,7\,8}{1\,2\,3\,4\,5\,6\,7\,8}$
.....
Gums .....

CHEST: Abnormalities.....
.....

LUNGS: Chronic Cough.....
Apical Fields, Right.....Left .....
Sputum .....Temperature .....
Microscopic Examination.....
Von Pirquet Test .....

HEART AND BLOOD VESSELS:
Location Apex Beat.....
Rhythm.....Rate.....Blood Pressure.....
Lesions .....
Condition Blood Vessels.....
Varicose Veins.....
Varicose Ulcers.....

ABDOMEN AND GENITO-URINARY TEST:
Abdomen: L. M. S..... Visceroptosis.....
Liver.....Spleen .....
Kidneys: Palpability.....Tenderness.....
Urine Examination: Specific Gravity....Reaction..
Albumin.....Sugar.....Microscopic Exam.
Hemorrhoids.....Fistula .....

HERNIA: Inguinal, Right.....Left.....
Femoral, Right.....Left.....
Umbilical .....

GENITAL ORGANS: Gonorrhea, active.....
Syphilis, active.....
Syphilis, history of.....Date.....
Chancroid .....
Hydrocele .....
Varicocele .....
Constipation .....
Diarrhea .....
Fits .....
Epilepsy .....
Fainting Spells.....
MUSCULAR SYSTEM:
Pain or restricted motion.....
Atrophies .....
GLANDULAR SYSTEM: Thyroid
Inguinal.....Cervical.....
Axillary .....
Cubital.....
SPINE: Scoliosis.....Kyphosis.....Lordosis .....
EXTREMITIES: Flatfoot: Right.....Left.....
Deformities or physical defects.....

Vaccination .....
Amputations .....
Skin Disease .....
Reflexes.....Equilibrium .....
HABITS: Liquor .....Yes..... No.....
Tobacco .....Yes..... No.....
Mentality: Bright.....Average.....Dull.....
Movements: Active.....Average.....Slow .....
Serious Operations, Illnesses and Injuries:
.....
.....
.....

GENERAL REMARKS AND RECOMMENDATIONS:
.....
.....
.....
.....
.....
.....
.....
Class.....
Examining Physician:

but one side of the question, and it is essential to realize that each large move in industrial medicine must have two results. It must aid the firm, and at the same time it must aid the employees. Lacking either of these advantages, the move does not contain those honest elements which make for

FIGURE 2

PHYSICAL EXAMINATION

Medical No.....
Dental No. .....
Employment No.....
Date .....

Name..... Age..... Sex.....
Dept..... Height..... Weight .....
Position..... Previous Occupation.....

Nutrition..... Anemia..... Tonsils .....
Bad Cough..... Glands..... Teeth .....
(See Dental Chart)
Hearing..... Sight R.....L..... Goiter .....
(Good, Fair, Poor)
Heart..... Lungs..... Hernia.....
Blood Pressure..... Hemorrhoids..... Urine .....
Albumin—Sugar
Operations (Age)..... Previous Illness (Note details on reverse side).....
Remarks or Additions:
.....
.....
.....

certain success. The attitude of labor in general toward physical examinations as a condition for employment has, in the main, been unfavorable. It will be remembered that one of the demands made during the steel strike of 1919 was the abolition of such examinations, the employees holding that the examination as then conducted was unfair to their interests. There is, however, abundant evidence that labor recognizes very fully the great advantages which thorough and fair medical service can supply. Thus, the organized garment trades in New York now require an extremely searching physical examination for admission to the union (2). An action such as this indicates at once that in the organization of a medical department in a store or in an industry of any type, effort should be made to give the employees a voice in the general control of the department, and in this way to cause removal of the possibility for discrimination, unfair use of records, etc., which the employees fear.

The character of the physical examination for employment varies markedly in industry. Figure 1 represents a form used in a large meat-packing establishment. It is obviously so long and complex that a great amount of time is required to fill it in carefully, and unless such record forms are adapted to the need they should fill and to the time which can be given to each candi-

FIGURE 3

MEDICAL EXAMINER'S REPORT

Rate of pulse ..... Posture ..... Does it intermit or become irregular? ..... Cause? .....

Character ..... Feet ..... Measurement of Chest: Inspiration ..... Abdomen at umbilicus .....

Height ..... Weight ..... Expiration .....

Race ..... General Appearance ..... Blood pressure .....

Arteries palpable? .....

After careful inquiry and physical examination do you find any evidence of past or present disease of:

Lungs or Pleurae .....

Brain or Nervous System .....

Heart .....

Blood Vessels .....

Skin .....

Nose and Throat .....

Stomach or Abdominal Organs .....

Gained or lost any weight in past year .....

Condition of teeth .....

Is hearing impaired? .....

Is eyesight normal? .....

Is hernia present? .....

Has he the mark of a successful vaccination? ..

REMARKS

URINE EXAMINATION

Specific gravity ..... Reaction .....

Albumin? ..... Sugar? .....

QUESTIONS TO BE ANSWERED IF APPLICANT IS A WOMAN

Married or single? ..... Is husband living? .....

Is she pregnant? ..... No. of children living ..... Age of eldest ..... Age of youngest .....

No. of children dead .....

Has she ever had a miscarriage? ..... Has she since had labor of full term? .....

Has she passed the climacteric? ..... When .....

Is menstruation regular? ..... Pain? ..... Reflex symptoms? .....

Signature .....M.D. Address .....

Medical Examiner

FIGURE 4

Accepted

Rejected

Accepted provisionally?

History No. .....

Date.....

Name of Applicant	Address	Department

Age	Place of Birth	How long in New York?

		AGE IF LIVING	STATE OF HEALTH	AGE AT DEATH	CAUSE OF DEATH	DURATION OF FATAL ILLNESS	To what extent does he use stimulants?
Father							
Mother							
Brothers	No. Living						To what extent does he use tobacco?
	No. Dead						
Sisters	No. Living						Other habits?
	No. Dead						

Have any of the applicant's uncles, aunts, or grandparents had insanity, cancer or tuberculosis?.....

Has he within one year occupied same room or house with a consumptive? .....

What physician did he last consult..... When ..... For what..... Duration of illness ..... Present health .....

What illnesses, diseases, accidents or surgical operations has he had since childhood? .....

Name of Disease, etc.	No. of attacks	Date of each	Duration	Severity	Results	Date of recovering

Signature.....

date for employment, they are worse than useless. Figure 2 is a form used by a large manufacturer of electric lamps, a firm employing many thousand men and women. It is brief but contains the essentials and may be filled in rapidly. Figure 3 is an example of a form now in use in a large department store. It is a very good one and possesses on the reverse side the medical history sheet shown in Figure 4. It seems probable that this store requires more than is necessary to gain good results.

After examining the store situation and bringing to bear upon it such elements of industrial medical experience as seem pertinent, we have designed the brief form shown in Figures 5 and 6. This may be made up on a 5 by 8-inch card. On the front are contained the necessary social data, occupational data, important past medical history, and recommendations to the employment department; on the back are the points covered by the physical examination. Such cards may be filed and to them may be clipped others of similar size upon which are recorded subsequent visits to the physician's office, their cause and treatment. The record kept of such visits should be as detailed as the condition warrants. A word or two will frequently suffice, but unless a rigid rule is made that no visit shall go unnoted, it is certain that sooner or later the store physician will awake to the consequences of having seriously neglected to notice the advance of chronic disease.

The technique of introducing the methods of a modern medical department in the store may be varied. If a decision has been reached to institute physical examinations for employment, due publicity should be given and the plans for work made clear to everyone involved. First, the executives should be informed and their approval and co-operation obtained; then the buyers, departmental heads, and supervisors should clearly understand the plans. Finally,

FIGURE 5

PHYSICAL EXAMINATION

Date of Examination.....

Weight.....Height.....Vaccination .....

Inoculation .....

Skin .....

Head.....

Eyes, R.....L.....

Ears, R.....L.....

Nose.....Throat .....

Teeth .....Neck and Glands.....

Chest .....Heart rate.....Size .....

Sounds.....B.P.....

Lungs .....

Abdomen .....

Pelvic .....

Extremities, Upper.....Lower .....

Feet.....Posture.....

Reflexes .....

Urine.....Blood .....

Comments:

General Appearance.....

Apparent Age.....Temperament.....

Peculiarities .....

Examined by.....

through the store paper or through a special bulletin, a concise and clear statement should be made to all the employees and, if an employees' organization exists, the fact that the medical department will be under a co-operative board of control should be especially emphasized. After all this has been done it will be found that success depends upon the personality and training of the physician in charge, and the qualifications which he or she should possess have been made clear in the first paper of this series.

Should the store desire a less vigorous course than this, it may well start by first requiring physical examinations of all food

handlers and elevator operators, for public safety is here directly involved. Next offering free examinations will bring volunteers. The character of the work done will then determine its success and scope. If, at first, attendance at the doctor's office falls off, no uneasiness need be felt. The success of the medical department depends far more upon the excellence of the work done than upon any other feature, and, given efficiency in this direction, the matter of attendance will soon take care of itself.

FIGURE 6

.....Store

Name.....Address..... M. or F., S.M.W.....

Age.....

Occupational History .....

Past Medical History .....

Recommendations to Employment Dept. .....

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A STATISTICAL STUDY OF ACCIDENTS IN THE COTTON MILLS, PRINT WORKS AND WORSTED MILLS OF A TEXTILE COMPANY*

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THIS paper deals with the statistical determination of the modes of occurrence of about 2500 accidents in a large textile company in New England, covering the calendar years of 1918 and 1919. The business of the company in question is the manufacture of cotton and worsted cloth, and the printing of cotton cloth. The company employs about 7500 people. The material for the study was compiled from the company's accident reports — reports made up in accordance with the provisions of the Massachusetts Compensation Act of 1911. Owing to the large number of accidents involved in the study, limits were made to the extent of the analysis as follows:

1. The recorded facts of the accidents were divided into groups:
   a. Hour of day of occurrence
   b. Day of the week
   c. Day of the month
   d. Age of the injured
   e. Sex of the injured
   f. Married or single
   g. Wage of the injured
   h. Time or piece rate

2. The lost-time accidents during 1919 were separated by departments. There were 213 cases of this sort.

3. The total of 2500 accidents was separated by departments.

4. An analysis of the results of this tabulation was made, methods of presentation of the tabulation to the groups interested were discussed, and a procedure for applying practically the results of the analysis, together with the organization for carrying them out, was outlined.

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The organization of the plant studied is as follows:

1. The print works is made up of twenty-four departments, which include the bleaching, dyeing, finishing and printing of cotton cloth. It is entirely separate from the other parts of the company's plants. About 2300 employees are on its payroll, this number being about 34 per cent. of the total in all of the mills. Of this number, about 1750 are male and the remaining 550 are female.

2. The worsted department is a separate division. The manufacturing process includes all operations from the raw wool to the weaving of the finished product. The employees number about 2800, with 1500 females and 1300 males.

3. The cotton department is also a separate division, but with transportation facilities closely allied to the print works. Here, out of a total of about 1200 employees, there are 650 females and 550 males.

4. The purchasing department is the necessary yard force and all of the transportation facilities among the various plants. This transportation is carried on by means of motor trucks. There are in this department about 400 employees, of whom 350 are males.

Classification of Accidents by Accompanying Circumstances

When an accident occurs, the Compensation Act requires that eight facts in regard to the circumstances of the accident and of the injured person be noted — namely, the hour of the day, the day of the week, and the day of the month on which the acci-

dent occurred, the age, sex, and marital condition of the injured person, his wage, and whether his work was time or piece work. When these facts are studied in connection with a large number of accidents, does a frequency-of-occurrence curve result of any practical value?

A. *Hour of the Day.* — From the figures in regard to the hours of accident occurrence the most striking frequency curve of the entire investigation is obtained. The accidents occur most frequently in all departments at the hours of 9 A.M. and 3 P.M. The question at once arises: What are the factors that would offer a solution? There are, first of all, the physical ones. These include:

1. The number of employees in the plant. Obviously there is no difference between these hours and any other working hours in the matter of number of employees.

2. Machines in operation. There is no reason to believe that there is an increase in the number of machines in operation during any particular hours.

3. The speed of the machines. There is no increase in this particular.

4. There is practically the same amount of sunshine at these hours during all seasons of the year.

5. The periods are equidistant from the last time food was taken. Of all the physical facts, this one is the most striking and will serve well to introduce the second set of factors which may offer a solution; i. e., the physiological ones.

The physiological factors are:

1. The physical condition of the employee at the time of the accident. Fatigue and its connection with the food-taking period produces a mental condition, dependent upon the physical one.

2. The age of the injured employee.

3. The sex of the injured person.

In order to discuss this question thoroughly, it will be necessary to anticipate the later sections of this report enough to say that the commonest age for the accident-person is 18 years and the predominant sex is male. Taken together, these facts as brought out by this inquiry lead up to the question: How does it happen that accidents occur most frequently to an 18-year old boy, at the hours stated above? For it is but reasonable to suppose that if this "peak man" can be dealt with, a real solution is possible.

The "peak man" is a person who is not only young, but has the most striking characters of quite immature youth. Such characters are a general mental attitude of carelessness and a very natural aptitude to be sleepy after eating. It is here that a difference of opinion will enter in. It is a question in my own mind as to whether this accident occurrence is due to carelessness from a certain mid-morning and mid-afternoon drowsiness or to the fact that that low mental period is passed and the fastest work of the day is being done. In either case there would occur a high accident rate, for were it a case of drowsiness, fingers would not be so swift to move out of danger as in the other periods of the day, or if it were a case of high speed work, coupled with the youthfulness of the employee, a willingness to go on with the work and "take a chance" on getting hurt might produce injuries. There is a good deal to be said, I think, for the idea that, especially in the morning, the high-speed work is the cause of accident. By high speed I mean the fact that the employee is working at his fastest rate and is doing everything that the task requires, whereas later the little niceties of the job are left to the realm of "I guess that will do." There is more attention to the more complete handling of the work in the freshness of the morning than there is later on in the day. And at the same time there goes with this completeness of work a closeness to the machine and its parts which would have more possibilities of accidents.

Granting that the after-dinner feeling does not extend till 3 o'clock, there would come a period of extra energy at this hour due to the very fact that a period of slow work had just preceded it. The accident hour at this time may represent an attempt to make up for the slow work done just previously.

In many parts of the textile industry, there is involved the work of supplying raw material for a machine which operates continuously. During a period of drowsiness, there would be a temptation to let the entire raw material for the machine run to a low point, with the result that a time of extra fast work to restock would be required. Take, for example, the four machines — a spinning frame, a loom, a worsted comb and a drawing frame — the operation of each of which is a continuous process. What would be more natural than for a drowsy operator to let the raw material supply "slide" for a longer period than usual after lunch and then have to be very active to fill up the frame? And in the loom, the question of supplying new bobbins would be put off after lunch for as long a period as possible.

Summarizing, it is my opinion that the theory of the high-speed cause for accidents will hold better for the morning than for the afternoon, but for reasons given above I should apply it to both times. In the afternoon, moreover, natural fatigue may more easily operate than in the morning.

B. *Day of the Week.* — The most striking fact brought out by a study of the day of the week upon which accidents occur is the large proportion of accidents during the forenoon on Saturdays. The average daily accident rate is 449 with a half-day average of 224+. But the half-day on Saturday shows an average of 265 accidents, which is 18 per cent. above normal. There are various factors which might influence this result, among these being the peculiar nature of the work done on Saturday, the mental processes of the employee so full of the plans for the afternoon that a natural carelessness would result, a rush to finish the week's work, and the necessity for cleaning and clearing of machines.

C. *Day of the Month.* — The chief fact brought out by this tabulation is of interest to the textile company studied rather than to the general public, and consists in the increasing accidents reported after July 1, 1919. This result is due to the emphasis placed by the safety engineer on the slightest injury in order to catch early accidents which have possibility of further trouble, such as infection from scratches. As safety work rests on the theory of accident prevention, it is essential that prevention start at the source of the trouble and that an injury be caught when it is still a minor one.

D. *Age of the Injured.* — The high mark of frequency is at 18 years. This fact is especially valuable as showing the youth of the injured and placing the greatest emphasis on accident prevention on this type of person. Were the figures available, it might be shown that one of the reasons for this "peak age" was the fact that of all of the employees in the plants the greater number were of this age. But even if this were the case, it is still of importance to know that so young an employee is the bearer of the greater accident risk.

E. *Sex of the Injured.* — Nearly 80 per cent. of the total accidents happened to males. Against this fact must be set the percentage of males in the plant. The figures and percentages are as follows:

Sex	First Part of 1918	Last Part of 1918	March, 1920
Males	4369....64%	4799....62%	4718....61%
Females ...	2352....36%	2926....38%	2922....39%

The peculiar nature of the industry has a good deal to do with these figures. The

women are employed in the parts of the mills where the lighter work is done. The men are employed where the more cumbersome machines are operated. The loom fixers, for instance, are men, and the work is done around the more crowded parts of the machine, often when the machine must be turned off and on. In general, considering the nature of the men's work it is no wonder that upon them falls the greater part of the injuries. The knowledge of this fact merely emphasizes the necessity for watching more closely those operations where men are largely employed and seeing that their machines and processes be more carefully and thoroughly studied with a view to eliminating as far as possible the accident risk.

F. *Married and Single.* — The figures upon this fact are nearly equally divided, a slightly greater number of injured persons being married. This is one of the main reasons for the benefits of the compensation act — to serve as a safeguard for the family during the enforced idleness of the injured husband or father.

G. *Wage of the Injured.* — Here the modal peak falls on the $18 man. The main question which might be raised in this connection would be in the application of the compensation act. With the working wage in such times as these only $18, what percentage of this wage had better be paid as compensation for lost time? Eighteen dollars per week represents a round sum of $900 per working year. How much less than this should be guaranteed in case of failure to be able to work? It would seem that at least two-thirds of this wage should be paid, if not three-fourths, in order to insure a reasonable income during disability.

H. *Time and Piece Work.* — The greater portion of the work in the mills, as shown by the compilation, is done on a straight time rate. This at once removes as a cause of accidents rush work being done on piece rates. Nothing of special value is to be learned from the figures on time and piece work. In a more detailed special inquiry, it might be well to have the accident frequency at some of the piece-rate machines charted.

This concludes a summary of the data obtained from classifying the eight facts in regard to accidents required by the compensation act. What, in the main, have been the outstanding benefits derived from this tabulation of about 18,000 facts as noted in the accident reports? We have learned that out of this group of accidents, each of which was apparently totally independent of the other, there appear certain more or less striking similarities in the circumstances surrounding them. In other words, we have become orientated for further inquiry. Methods of attacking the peaks of accident occurrence may differ, but it is clear that there must be agreement in the goal sought for.

Whatever the cause of accidents, undoubtedly one of the best ways to attack the problem of their prevention is to present the facts clearly to the operators and to arouse their interest in the problem. This should be done through verbal and picture instruction. Nothing lends itself to graphic presentation better than this matter of accident frequency. The facts of accident occurrence may be presented either in the form of a straight bar chart or in the better form of a frequency curve on a clock base. Perhaps in this connection I had best say a few words as to the general policy of presenting graphs to great numbers of employees. In the first place the graphs must be striking. The eye will not take cognizance of a small difference. This point is well illustrated by the use of a graph presentation of the hourly accident rate and the daily accident rate. In the former case, there is a wide difference in the number of accidents which causes the hours to be marked off clearly, while in the latter

case there is at best only a little less than one-fifth difference in length of the two average bars. A frequency at least double the other frequencies presented should be present to justify the use of a graph. In the second place, a graph should be simple. It

TABLE 1. — COMPARISON OF PERCENTAGE DISTRIBUTION OF ACCIDENTS IN THE TEXTILE INDUSTRY WITH ACCIDENTS OF INDUSTRY IN GENERAL

		2500 Accidents in Textile Industry	Mass. Accident Board
Sex of the Injured	Male	79	91.3
	Female	21	8.7
		100%	100%
Time or Piece Work	Time	88.3	86.7
	Piece	11.7	13.3
		100%	100%
Wage Distribution	Up to $8	0.4	6.7
	$8 to $15	18.5	55.8
	$15 to $20	53.1	24.5
	Over $20	28.0	13.0
		100%	100%
Age Distribution	Under 16	6.7	1.8
	16–20	15.8	13.3
	21–29	25.9	33.9
	30–39	20.3	23.7
	40–49	17.4	15.7
	50–59	9.6	8.3
	Over 60	4.3	3.3
		100%	100%

should contain the minimum of subjects and the lines should be clear cut and not mixed or crossed too often by other lines. Popularity will lie along this same line of simplicity. A graph using hours of the day should be made up in the form of a clock. Graphs of temperature should be modeled after a thermometer. Two graphs, moreover, should rarely be shown together. The mental picture which results, like a photographic plate duplication of objects, is apt to blur.

In the *Fifth Annual Report* of the Massachusetts Industrial Accident Board, for the year ending June 30, 1917, the percentage distribution of total accidents is given by groups. For purposes of comparison, I have drawn up a similar distribution of the accidents entering into this study (Table 1). In a comparison of these figures, it must be kept in mind that in the textiles we are dealing with one special industry and are comparing its figures with the percentages drawn from general industry. If for no other reason but for emphasis on the special characteristics of the textile industry, these figures are of value.

The character of the textile industry is shown especially well in the first and last comparisons in Table 1. The percentage of females is considerably higher in the textiles than in general industry, as the percentage of accident occurrence shows. Also the percentage of young persons is higher in the textiles than in other industries, as shown by the differences in percentages of those under 20 years in the above figures. The shift in the wage distribution groups is caused by the general rise in the rate of wages during the last two years. For the greater part, there seems to be a system of day wages in the textiles as in any other industry, or, speaking from an accident point of view, the number of accidents due to speed caused through a piece rate are no greater in the textile industry than in any other.

Occurrence of Accidents by Departments

In this section comparisons will be made between the lost-time accidents and the total accidents, both of which have been divided and allocated by departments. In the tables submitted with this section, a graphical presentation of the accident occurrence is shown, but for purposes of the greatest utility only those departments showing the relatively highest accident rates will be discussed. For it has been a principle of this report to deal only with the occurrence and treatment of the peaks

of the accident problem. In this discussion the purchasing department will be omitted as it is a single and special department in itself and entirely independent from the more technical industrial processes.

In the table which is given below (Table 2) the top accident departments are selected for examination. From these figures some idea of the relation between the lost time and the total accident rate may be gained. The italicized names appear among the highest rate accident departments in the figures both for lost time and for total accident occurrence. Of the seventeen names on this list, seven appear in both columns. There is a total of fifty-six overseers in these plants. If the accident rate were the same for each overseer, with an average of 2500 accidents and 213 lost-time accidents, each overseer would have as his rate 3.5 lost-time accidents and 44.5 total accidents. This compilation shows how much above this hypothetical rate the "high peak" overseers average. Out of 2500 accidents, these seven overseers had in their departments 771 accidents. Numerically stated, this means that one-eighth of the overseers had one-third of all of the accidents. In the same manner, these overseers had a little less than one-third of all of the lost-time accidents.

What is the nature of the work in these departments to cause such a high accident rate? To aid in this answer, these departments may be grouped as follows:

Worsted	Through drawing (Walden) Repair (James)
Print Works	Repair (Jackson) Printing (Albee)
Cotton	Through roving * (Simmons) Spinning (Brink)

This grouping shows certain rather striking facts. In the case of cotton manufacturing, the groups show that whatever methods of accident prevention have been adopted, there is still a great deal to be done. For in cotton, the accident frequency is greatest in the industry throughout the entire manufacture of the spun thread.

TABLE 2. — CLASSIFICATION OF ACCIDENTS BY DEPARTMENTS

Lost-Time Accidents		Total Accidents		
Worsted Mills				
53.9% of lost-time accidents in worsted mills	*Walden* 13	*Walden* 116	47.4% of total worsted mills accidents	
	James 8	*James* 158		
	Hastings 13	Sturgis 90		
	Noble 7	Childs 81		
	41	445		
Print Works				
42.6% of lost-time accidents in print works	*Jackson* 8	*Jackson* 142	43.1% of total print works accidents	
	Kingman 8	*Kingman* 70		
	Albee 6	*Albee* 122		
	Shaw 7	Atkinson 81		
	Taylor 6	Burns 93		
	35	508		
Cotton Mills				
51.7% of lost-time accidents in cotton mills	*Brink* 10	*Brink* 80	57.8% of total cotton mills accidents	
	Simmons 10	*Simmons* 83		
	Rowley 9	Warner 63		
	29	226		

This series of processes includes the entire machine list up to the looms.

In worsted manufacture, the fact of the longer staple of the raw material may influence the accident rate. The fact that there are less interruptions to the process of manufacture due to a breaking of the fabric in its passage through the machines will probably be responsible for the less degree of risk in the spinning process as compared to the cotton.

The fact that the repair department should appear twice is more easily explained. This department is a peculiarly risky one due to the fact that it is work

* Through roving means that these departments include all the processes from the picking and carding machines in the manufacturing process to the time when the material is presented to the spinning machines.

demanding much closer association with machines out of order, and where the risk arising from accidental starting and stopping of the machines is greater. The men in these departments are constantly doing work on the more complicated parts of the machines, work requiring frequent starting and stopping of the machines to determine whether or not the repairs are satisfactory. Moreover, a greater number of machine tools are being handled in these departments than elsewhere.

In the printing process, there are two major causes for the higher accident rate. One of these is the "doctor-blade," an instrument used in connection with the printing machine which has an unduly large number of accidents. This blade is a long one used for scraping off the surplus of color from the copper rolls as they turn against the print cloth. This blade is very sharp and requires frequent sharpening by a hand method. In running over a list of the accidents in the print works, I am surprised at the number of reports which state that the employee was cut on this blade. The process of sharpening is identical with the ordinary method of a boy sharpening a jack-knife, but here the blade is much sharper and at least 36 inches long. The smoothing off process after the actual sharpening is particularly conducive to accidents because of the danger from cuts when the sharp edge of the long blade is rubbed with the smoothing cloth.

In this discussion of the reasons for accident disposition I have not attempted to cover all of the causes of accidents, but I have attempted to bring out the most salient features of accident occurrence in the textile industry. The main purpose of this study will have been achieved if the engineer has been guided in his work to the important points of attack in the reduction of accident occurrence. For at this point, the work of the statistician must end; actual accident prevention is a process of safeguarding machines against the possibility of accidents, and this is the engineer's task.

INDUSTRIAL "GASSING" AND THE EDWARD MEDAL*

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WE have been thrilled during the war by deeds of bravery done by the highest and lowest in the army, and no less do those who know the risks run by workers in munition factories at home stint praise of the presence of mind, courage, and self-sacrifice shown by workers in attempting to extinguish fires or prevent explosions, or in risking health in the service of their country. This, however, is no new thing; and when I look through the reports on cases of poisoning by fumes and gases, I am always carried away, as in the reading of a romance, by the excitement of the event, the tangled thread of circumstance which has brought the situation about, the attendant sins of omission and commission, and I breathe a sigh of relief or feel a lump in the throat, as success or disaster attends the effort at rescue.

When we talk in cold blood about workers, reference is sometimes made to traits of weariness that come over them, as evinced in limitation of output. We speak of "Weary Willies" and of a "Ca' Canny" policy. But where a workman's life is in danger, I have yet to read of any fellow worker shirking the effort at rescue, or even hesitating to risk his own life in the attempt. The scenes thus visualised carry the mind's eye to high gantry stagings — the feeding places on the tops of blast furnaces — to cleaning operations inside retorts — acid eggs — tank wagons, access to which has to be gained through manholes so small that a man must squeeze his body to pass through, to dark underground passages in the mine where, crawling on his hands and knees in mephitic atmosphere, and running in danger from falling roofs, the rescue party slowly makes its way. The whole atmosphere around these gassing cases is tense in the highest degree. Even the language in which the inspector describes the occurrence has in it something of the knell of fate:

> During salvage operations on board the steamship *Great City*, the holds of which the men knew to be heavily charged with gas arising from decomposing grain, one of the stevedore's men noticed some pieces of wood floating towards the pump and, contrary to strict orders, went down to pick the wood up so as to prevent the pump from choking. While in the act of doing this, he was overcome by gas and fell into the water. Anderson, who had some time previously suffered from gas poisoning, and therefore knew the gravity of the risk at once went to his assistance and succeeded in holding him up while a rope was being sent down to him, but before this could reach him he was also overcome and fell into the water. Tierney and Gab then went to the rescue, but both were also overcome. Anderson, Tierney and Gab undoubtedly lost their lives in an endeavour to save the lives of their fellow workman!

The following are instances of the dramatic nature of carbonic oxide poisoning, of which the symptoms are so insidious — almost the first effect being to rob the workman of the use of his limbs:

> A man named Heald descended into a melting furnace 30 feet high in order to adjust a piece of scrap iron under which some coke had to be placed. Heald descended into the furnace by a chain, and was immediately overcome by noxious gas which had collected there. A man standing at the top at once gave the alarm and Shadrach Jackson and Ince came to his assistance. Jackson without hesitation slid down the chain, and a rope was thrown to him, which he tied round Heald, who was then pulled up, Jackson climbing up the chain after him. As Jackson, however, reached the door through which Heald was being pulled he too was overcome by the gas and fell back to the bottom of the furnace. Ince then descended and placed a rope round Jackson's body, but unfortunately it slipped off before the men could

* Lowell Institute Lecture, delivered in Boston, Mass., Dec. 4, 1919. Received for publication Feb. 1, 1920.

be pulled out, though Ince, feeling that he was overcome by the gas, climbed up the chains and escaped. On recovering, Ince went down a second time, once more tied the rope round Jackson, but again failed to bring him to the surface, though he himself escaped by a ladder which had been brought. At this, Darley then went down the ladder with a rope, which was attached to Jackson's belt, but unfortunately the belt gave way. On Darley coming up, Speke went down, and the rope being more securely tied round Jackson's body, he was extricated, though unhappily, on reaching the surface it was found that he was dead. All the men were fully aware of the risk towhich they repeatedly exposed themselves.

How could those men have known that one of the first effects of carbonic oxide gas would be to rob them of the use of their limbs, and how comes it that no mention is made of the use of a safety helmet or mask?

Every year carbonic oxide gas claims more victims than any other gas in factories and workshops because of the wide extension its use has undergone in the manufacture of water gas, and of other gases of a similar nature — suction gas, Dowson gas, Mond gas, power gas, producer gas, and blast furnace gas for driving gas engines, heating furnaces and boilers, welding, soldering, and other industrial purposes. Whereas the proportion of carbonic oxide in coal gas varies from 4 to 12 per cent., in carburetted water gas it reaches 30 per cent., and in blast furnace gas about 25 per cent.

The casualties are traceable to several common causes, of which the principal are, besides ordinary leakages from joints and taps or in pipes or flues conveying gas: (1) gradual escape into small engine rooms, weigh cabins or offices; (2) cleaning and repairing engines and tanks, scrumbles or blast furnace flues; (3) inefficient disconnection from the blast furnace of the flue during cleaning; (4) charging cupola furnaces; (5) percolation of gas through several yards of soil from underground flues; (6) conveyance by the wind of gas escaping from defective reservoirs through ventilators and open windows into workrooms at a distance; (7) ignorance of the danger and of the earliest symptoms; (8) inodorous nature of the gas; (9) working alone; and (10) lack of rescue appliances.

A workman employed by the Frodingham Iron & Steel Co., Ltd., was overcome by gas in a manhole at the Company's blast furnace at Scunthorpe. After several ineffectual attempts had been made to rescue the unfortunate man, Thackeray, a fellow workman, volunteered and, tying a handkerchief over his mouth, entered the manhole and succeeded in raising the man sufficiently to enable other workmen outside to get him out, but it was too late to restore him. Thackeray himself was overcome by the gas and did not recover entire consciousness until the following morning.

Tied a handkerchief round his mouth! Again ignorance of the danger of the symptoms and of the right means of rescue!

An insidious gas of which you do not require to be told the poisonous properties — sulphuretted hydrogen gas which, in a proportion of 1 in 1000 parts of air, causes unconsciousness — is a source of poisoning in the cleaning out of tar stills and of sewers in the absence of proper precautions.

Alfred Butterfield, a workman employed at the Luton Corporation descended a sump hole at the Luton Sewage Farm and was overcome by foul gas which had accumulated there. William Brandon immediately went to his rescue and descended the hole but was overcome by the gas. In spite of the serious danger disclosed by the previous attempt at rescue, Herbert Carter decided to make the descent and placing a handkerchief over his mouth, was lowered by two ropes into the hole. There he found that Butterfield was dead and Brandon was unconscious. He placed a rope round Brandon and they were both drawn to the surface. Carter then descended again and attached a rope to Butterfield.

A handkerchief again! You will almost think it is the only remedy we can suggest in England. Such is, however, not quite the case. The measures we recommend in cleaning out tar stills are: (1) complete isolation from adjoining tar stills; (2) ventilation of every tar still before persons are

allowed to enter; (3) inspection by the foreman of the tar still before any workman is allowed in it; (4) foreman and all persons employed in tar stills should wear a rope securely fastened, with a long end left free, and two men outside whose sole duty should be to watch and draw out any person appearing to be affected; (5) provision of an oxygen cylinder with a mouth piece and printed instructions as to the use of the bottle and the method of employing artificial respiration.

The cases I have cited are all instances of the award of the Edward Medal which, on the recommendation of the Home Secretary alone, the King, as representing the country, has made since 1909 to his subjects who, in the course of industrial employment, risk their lives in the effort at rescue not only in Great Britain, but in all British Dominions or territories under his protection. The medal bears on the reverse the King's effigy, and on the obverse a design and the words "For Courage."

I thought before I came to the States that you had nothing quite comparable with the Edward Medal. Neither have you, but I had forgotten the Carnegie Hero Fund, the records of the awards under which tell just the same story, with even worse insistence — ludicrous indeed were it not so tragic — of the fatal pocket handkerchief. Thus:

Edward Davis, 32, died saving James Dougherty on March 27, 1916. With a wet handkerchief over his nose and mouth he entered the kettle full of deadly fumes, but he was affected by the fumes before he could lift Dougherty, and hurriedly ascended. After another man had made an unsuccessful attempt, Davis re-entered with the handkerchief over his face and a rope tied round him. He tied another rope to Dougherty and hurried from the kettle. As he emerged he gasped and fell unconscious, and efforts to save him with the pulmotor were fruitless.

Perhaps of all the gases and fumes the most treacherous are nitrous fumes. They are commonly given off in nitrating processes in explosive and synthetic dye factories. They seem at any rate to be the fumes with which familiarity breeds contempt more readily than with others, and with them perhaps the folded handkerchief is the rescue appliance relied on more often than any other. The full effect of inhaling these fumes is not felt immediately, and so workers often continue at work and unwittingly inhale a fatal dose. What happens then is that after the initial gassing effects pass off, the workman feels better and continues his work for perhaps the rest of the day. The increased exertion of going home makes him feel short of breath, and this shortening of the breath gets steadily worse until he swims for his life. The explanation of the delayed action is simple, seeing that it takes a few hours for the inflammatory process set up in his lungs to develop, and finally to drown him in his own secretions.

A man named Richard Morgan was repairing an electric wire on the top of a four-story building, when he was overcome by poisonous fumes given off by an overflow of acid on the ground floor. The fumes, which were dense and suffocating, soon filled the building, and McNab, a foreman fitter, and two other workmen went up an outside staircase provided for cases of emergency in search of Morgan, but receiving no reply to their shouts, descended. McNab then, hearing from other workmen that Morgan was on the roof, went up the staircase again, but without success. McNab then went up for the third time, and succeeded in entering the room where Morgan was. Crawling on his hands and knees, he managed to grasp Morgan's hand, and drag him out to the landing, where he obtained assistance in carrying Morgan downstairs. McNab showed presence of mind and persistent bravery in face of danger, though his effort to save Morgan's life was unfortunately unavailing, as Morgan succumbed after some hours to the effects of the poisonous fumes.

In addition to the resourcefulness and bravery shown in rescue work, perusal of the gassing cases bring out another equally significant feature too little dwelt upon — namely, the extraordinary sense of responsibility shown often by the workman and

the marvellous pertinacity and devotion to duty he displays, often in the face of dire peril to his own life. Instances I have cited show this and show how this sense of responsibility fôrces him even to break through the strictest orders. A man, Charles Smith of Rawtenstall, last year was in charge of some revolving benzine degreasing vats. Blow-off cocks were fitted, and one of them gave way, allowing the benzine to escape, of which there were 3000 gallons under a pressure of 30 pounds. Smith entered the vat room four times in unavailing attempts to turn off the steam. He was three times fetched out, overcome by the benzine fumes. After the third time he was left alone under a promise not to return, by the night foreman who was called away to search for two other men. On coming back he could not find Smith where he had left him and guessing what had happened he returned to the vat room to find him lying unconscious — unconsciousness from which he never recovered. Cases like this where death claims its victims come to light, but they are but instances of what happens more often than one can count. The paramount instinct of setting the wrong right — perhaps of correcting a mistake and being rid of the reproach of conscience — is there.

How can the way in which gases and fumes act be best presented to the mind's eye so that they do not confuse, but fall into their place in ordered sequence; so that, knowing the type, we can predict the probable action of a similar, related compound. Such a classification is not easy because classification should take account of the relative toxicity of the compounds — the fact that one is very poisonous, and another only slightly so, like the compounds of arsenic. Account must also be taken of the various processes and the way in which they may be used as, for example, the ease or otherwise with which exhaust ventilation can be applied; and if the list is not to lead to false ideas, it must be complete, or else omission may lead to the assumption that those omitted are harmless. This, for instance, has occurred in the case of benzol. In 1915 the French government wrote, asking for our experience of danger attaching to the use of benzene. They did not say for what purpose they intended to use it, but it turned out it was as a solvent for india-rubber in the proofing of balloon fabric. At that time, we had only had experience of unconsciousness produced by accidental breakage of a benzol still, not of chronic poisoning. But at the back of my mind I knew of one or two isolated instances of profound disturbance of the blood resulting in symptoms resembling scurvy, which had been reported in the United States and Sweden. I said to myself, "Is not this a negligible danger to which I had better not refer?" Well, those very symptoms of chronic benzol poisoning, a form of pernicious anemia, occurred later, and disturbed profoundly the peace of mind of the great rubber manufacturers in the country.

As a result of dilution with air, most industrial poisons can be absorbed only in very minute doses, yet some are so much more potent than others that one, like naphtha, may be disregarded, and another, like arseniuretted hydrogen gas or sulphuretted hydrogen gas or carbonic oxide, prove fatal. For some of them, therefore, the term industrial poison is a misnomer until the amount absorbed has reached a certain concentration. But we must have a classification, and the best line to adopt is a chemical basis, because whatever differences there may be between different members of the same chemical group, will be differences of degree and less marked than those produced by another chemical group. Thus, you would have the two main divisions of (1) INORGANIC, and (2) ORGANIC poisons.* The sub-divisions of (1) inorganic

* This list is practically that of Fischer, formerly Director of the Institute for Factory Hygiene, Frankfurt a. M.

poisons are: (a) *non-metallic* — chlorine, calcium chloride, hydrochloric acid, potassium chlorate, hydrofluoric acid, carbonic oxide, phosgene, carbon dioxide, cyanogen compounds, ammonia, nitrous fumes, phosphorus, phosphoretted hydrogen, arsenic compounds, antimony compounds, sulphur dioxide, sulphuric acid, sulphuretted hydrogen, carbon bisulphide, chloride of sulphur; and (b) *metallic* — chromic acid and chromates, manganese dioxide, sulphate of nickel, mercury and lead. The sub-divisions of (2) the organic substances are: (a) the *unsaturated carbon compounds* — benzene, petroleum, methyl, ethyl, amyl, and allyl-alcohol, oxalic acid, formal- and acetaldehyde, acrolein, acetone, methyl-bromide and iodine, nitroglycerine, dimethyl-sulphate and amyl acetate; and (b) the *aromatic series* — benzol, the nitro and amido derivatives of benzene, toluene and their homologues (which would include dinitrobenzene, trinitrotoluene, anilin, paranitranilin, etc.) and their chlorine substitution compounds. Then would come phenol and its nitro derivatives (dinitrophenol and picric acid), the chlorine compounds of carbon, ethylene and ethane (such as carbon tetrachloride and tetrachlorethane), and pyridin, naphthalin, benzidin, acridin and turpentine.

But we can summarize the effect on the tissues of various poisons briefly by classifying them as having action by:

1. Irritating the mucous membrane — chlorine, nitrous fumes, sulphur dioxide, formaldehyde.

2. Altering the constituents of the blood, which is peculiarly the property of the nitro and amido derivatives of benzene, of arseniuretted hydrogen gas, and of carbonic oxide gas.

3. Affecting the brain and nervous system — carbon bisulphide and the unsaturated carbon compounds.

4. Affecting metabolism by remote action on organs and tissues — phosphorus, lead, tetrachlorethane, benzol, toluene, and their nitro derivatives.

You see how complicated the matter becomes in attempting even so simple a classification as this, since overlapping comes in very quickly. When all is said and done, the number of the common industrial poisons is small, and you have done your duty if you have mastered these — carbonic oxide, sulphuretted hydrogen, lead, mercury, nitrous fumes, and arseniuretted hydrogen gas.

[Lantern slides of the Edward and Albert Medals, rescue appliances, and method of exhaust ventilation were shown.]

EXPERIMENTAL TRINITROTOLUENE POISONING*

SAMUEL R. HAYTHORN, M.D.

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IN October, 1917, an employee of a large trinitrotoluene (trinitrotoluol, T.N.T.) factory was admitted to the Allegheny General Hospital with an acute toxic jaundice. He lived less than twenty-four hours and the principal lesion found at autopsy was that of a very pronounced yellow atrophy of the liver. Reference to the literature showed that several such instances had been noted in England (1) (2) (3), and that one similar case had been reported (4) in the United States. In spite of the fact that the reports on animal work were very meagre (5) (6) (7) and were chiefly of the negative sort, we began at once to try to produce trinitrotoluene poisoning experimentally. We hoped not only to be able to study the histogenesis of the liver lesions in this way but also definitely to determine the most important route of entrance of the toxic substances to the body. Reports of our findings in the case of toxic jaundice and of our earlier experimental work were made in former papers (8) (9). The purpose of this paper is to report all of our experimental work, and to apply the results as far as it is possible in interpreting the clinical manifestations of T.N.T. poisoning.

Experiments to Determine the Most Important Route of Absorption of Trinitrotoluol by the Body

Trinitrotoluene workers are exposed to T.N.T. in several different ways, depending largely on the department of the factory in which they are employed. If the individual works in the nitrating room, he inhales gases from volatilized T.N.T. and from boiling acids. If he is in a grinding department or some other dusty part of the works, he gets a certain amount of it into his nose and mouth, on his hands, and into the meshes of his clothing, and probably inspires a small amount of it. If he is a packer of wet T.N.T., he is exposed to the dust adhering to the walls of the containers which are returned to be refilled, and, at times, also has his hands in contact with the wet, soggy, irritating masses of T.N.T. It appeared then, from the way in which the worker is brought in contact with T.N.T., that there were at least three possible routes of entrance for the poison; namely, through the air passages, through the skin, and by way of the alimentary tract.

Absorption along the Air Passages

Theoretically, there are two possible ways of producing poisoning by way of the air passages — one, by subjecting the animals to the fumes of volatilized T.N.T., and the other, by forcing the dust into their lungs.

Our first set of experiments was with the fumes and consisted in placing four guinea pigs in a closed compartment where they were exposed daily to the gases formed by the evaporation of T.N.T. The exposures at first made were of one-half hour's duration, but as this caused the animals to exhibit no symptoms, the time was increased until they were given about three hours exposure daily. At first, the fumes were generated by placing a container full of T.N.T. on an electrically heated water bath, in which the water was kept boiling. The animals were far more seriously affected by the steam than by the fumes, so that later an electric hot plate was substituted for the water bath and the T.N.T. powder sprayed upon the plate from a small atomizer.

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With this method, volatilization of the powder occurred instantly, so that the box was soon filled with yellow fumes. After about a month, the four animals were accidentally overcome by the heat in the box and died from the effects. These animals exhibited no lesions which could be ascribed to T.N.T., and as no uncontaminated samples of urine were obtained during life, and as their bladders were empty at autopsy, it is not possible to state whether or not they absorbed any T.N.T.

The second set of experiments was conducted on two guinea pigs and two rabbits. These animals had gags placed in their mouths so that the interference to inhalation due to the intricate turbinate bones was removed. The fumes were generated by the atomizer and hot plate method, but were led into the box through a short flue to prevent over-heating of the animals. Catheterized specimens of urine from the rabbits were examined daily. On the fifth day a positive Webster's test was given by the urine from one rabbit. All of the other findings in this group were negative.

Smith (10) made the observation, in discussing the above experiments, that when T.N.T. vaporizes it is broken up into several different gases so that the animals did not receive T.N.T. as such. The single positive finding can be explained by the absorption of the T.N.T. dust which sublimated on the skin of the rabbit.

We found no evidence that fumes produce T.N.T. poisoning, though they undoubtedly play a part in the condition discussed under the heading, *Acute Gassing*.

In a recent report on the investigation of T.N.T. poisoning by the United States Public Health Service (11), mention is made of the successful introduction of T.N.T. dust into the lower air passages. Moore (5) and Putnam and Herman (12) have shown that the quantity of T.N.T. dust which is present in the T.N.T. factory is so small that it is hardly likely to be the cause of the poisoning. We performed a few experiments in which we attempted to insufflate the lungs with T.N.T. dust by blowing the fine powder from an atomizer into the back of the pharynx, the current being continued long enough to force the animal to inspire deeply. Again, our method of feeding, which we will describe later, undoubtedly led to a deposit of some of the powder in the larynx and trachea. Large phagocytic endothelial leucocytes filled with bright yellow granules were found in the lungs of many of the animals. These granules did not give the iron reaction, and we attempted to apply a modified Webster's test to frozen sections while watching for the reaction under the microscope. Our tests failed to prove that these granules were T.N.T., but it is possible that they were.

We believe from the above cited evidence that it is possible to force T.N.T. powder into the lungs under experimental conditions, but that the route as a source of poisoning to the T.N.T. worker is an unimportant one.

Absorption through the Skin

Most clinical observers believe the skin to be the most common means of absorption of trinitrotoluene and it undoubtedly plays a considerable part in bringing about poisoning. However, some of the evidence which has been advanced to prove that the skin is the only important means of absorption is not entirely convincing. Moore's experiment upon himself has not been borne out by others, and I have rubbed large quantities of T.N.T powder on my arms for several days consecutively, have obtained a reddening of the skin from the procedure, but have been unable to produce a positive Webster's test. Putnam's and Herman's argument that the dirtiest men were always the sickest scarcely rules out the possibility of these individuals

having carried a larger amount of T.N.T. powder to their mouths by way of the hands, food, etc., than the other individuals did, and the argument that the protective varnish cut down the occurrence of the poisoning does not take into consideration the fact that this varnish was regularly removed by thorough washing and cleansing with alcohol before the men went to their meals and homes.

Experimentally we produced liver lesions of the red, hyalin type in guinea pigs by inunctions with 10 per cent. T.N.T. ointment, in which lanolin was used as a menstruum. The symptoms in these animals did not appear until after repeated inunctions, and positive Webster's tests were obtained on catheterized samples of urine from rabbits which had been rubbed between the shoulders with the ointment no earlier than five days after the first rubbing. Subcutaneous injections of T.N.T. mixed with lanolin (10 per cent.) produced positive Webster's tests in the urine. One young guinea pig died within twenty-four hours after a 2-c.c. subcutaneous injection of the material. This animal developed a general edema in which the fluids of the body were stained bright yellow. Five other animals injected on the same day showed no ill effects.

Absorption along the Alimentary Canal

From the standpoint of practical work it is scarcely possible to exclude the intestinal route. The workers complain of the bitter taste in the mouth and the substance is exceedingly soluble in saliva and in gastric juice. The use of "chewing tobacco" is a common habit among the employees, and before extreme cleanliness was insisted upon, the men were as a rule careless about washing well before eating their meals. The dust which collects in the posterior naso-pharynx is commonly swallowed. Altogether there are many ways in which T.N.T. may reach the intestinal tract.

Experimentally, feeding is the surest way of producing poisoning in animals, though individual susceptibility makes it very difficult to determine the lethal dose. Young guinea pigs are much more susceptible than older ones, but the latter occasionally show surprising variations. T.N.T. is quite soluble in milk and poisoning can be produced by feeding milk saturated with the powder. Graham crackers soaked in T.N.T. and milk produce very violent diarrheas in the animals, but the bitter taste makes it difficult to get the animals to eat them. We were most successful in depositing the powder in the back of an animal's throat and following this either with a drink of milk, water, or 10 per cent. alcohol, and holding the nose until the animal swallowed. Guinea pigs fed in this way presented poisoning symptoms in from three days to two weeks, depending on the dose, age of the pig, and individual susceptibility. Rabbits fed in a similar way showed blood changes and positive Webster's tests in catheterized samples of urine, after two or three feedings.

We conclude, therefore, from our experimental results that both the skin and alimentary tract are important routes of entrance of trinitrotoluene to the body, and that the lungs and air passages absorb apparently negligible quantities.

Solubility of T.N.T. in Body Fluids

We found that all of the body fluids which we tested would take up enough T.N.T. to give positive Webster's tests, but we gave up the endeavor to determine quantitative solubilities because of the difficulties of quantitative determinations, since it required widely varying quantities of different samples of the same fluid to absorb a given quantity of the powder, and because the value of the results did not ap-

pear to justify the work necessary. Several samples of each of the body fluids mentioned in the following table were incubated with T.N.T. powder for twenty-four hours, were filtered, the reaction taken, the general appearance noted, and the filtrate divided into two portions. One portion was examined by superimposing a layer of 40 per cent. potassium hydroxide in absolute alcohol; the other portion was acidified by sulphuric acid, extracted with ether, and tested with the hydroxide and alcohol solution according to Webster's method. The number of plus signs indicates roughly the relative rapidity and strength of the reaction and the permanency of the brown color which remained as the end reaction.

Body Fluid	Appearance	Direct Test	Webster's Test
Saliva	orange-brown	+	+ +
Stomach juice	colorless	+ + +	+ + +
Milk (cow's)	yellow to orange	+ + +	+ + + + +
Human bile	brown to black	+ + +	+
Human serum	dark brown	+ + + + +	+ +
Urine	yellow-brown	+ + +	+ + + +

Clinical Types of the Disease with Reference to Experimental Data

Trinitrotoluene in man has manifested itself in several different clinical forms, but it is not possible to differentiate all of them experimentally. The types generally described are "Acute Gassings," "Dermatitis," "Minor Poisonings," "Severe Toxemias," "Toxic Jaundice and Acute Yellow Atrophy of the Liver," "Toxic Anemia" and "Aplastic Anemia."

Acute Gassings. — This form of poisoning is due to the inhalation of poisonous gases formed during the manufacture of T.N.T., though not due to T.N.T. as a chemical entity. Dr. F. W. Matthewson (13), physician for a large T.N.T. factory, treated about seventy-five employees, who had been "gassed" while at their work. Most of the instances occurred in the nitrating room where toluol, sulphuric acid and nitric acid were mixed together and boiled in large vats called nitrators. The nitrators used in this plant were large vats 12 feet high and 10 feet in diameter, and were closed except for a small opening near the top through which the operator watched to see that the acids were boiling properly. The boiling was continued for two or three hours at a time and during this time the operator was more or less constantly exposed to acid gases. The bulk of the gases were carried away from the nitrators in iron pipes, which led into terra cotta pipes laid on lead sheeting to prevent acids from dripping through. According to Smith (10) there are always relatively large quantities of nitric acid, nitrous oxide, methane, hydrogen, and chlorine gases present in the nitrating rooms. Most of the instances of acute gassing followed sudden exposure to large quantities of gas, resulting from the erosion of pipes, minor explosions, or difficulty in controling the nitrator, but sometimes the clinical picture of acute gassing resulted from prolonged exposure to small quantities of the fumes.

The suddenly gassed individuals became choked, cyanotic and sometimes lost consciousness. They were resuscitated without much difficulty and given forced inhalations of aromatic spirits of ammonia which was kept on hand in iron cylinders. The men were generally removed to their homes in a few hours. The more severely gassed began to spit blood at once and some continued to do so for several days. Occasionally, the gassings were followed, within twenty-four hours, by a chill, and numerous instances of severe bronchitis and moderate edema of the lungs followed. Complete recovery after several days rest in bed was the rule. Some, however, developed bronchopneumonia, and there were two instances of lobar pneumonia.

A number of sputa from the bronchitis cases were sent us, and bacterial examination revealed the usual respiratory flora, with a predominence of pneumococci, streptococci and staphylococci. The bronchitis and bronchopneumonia apparently resulted from the implantation of these organisms on the injured surfaces of the bronchi and bronchioles. Wagner (14) reported an autopsy on a case of bronchiolitis obliterans in a T.N.T. worker. The lesions in his case probably resulted from an acute gassing which destroyed the superficial layers so that attempted healing took place by organization. Dr. J. P. McKelvy called my attention to a case of bronchial affection following prolonged exposure to gases in the nitrating room, which was of unusual interest from the radiographic findings.

The patient, when admitted to Dr. McKelvy's service at the Allegheny General Hospital, gave the following history. He had been employed in the nitrating room of a T.N.T. plant for a number of weeks, where he worked twelve hours a day in the fumes. For a month before his entrance to the hospital he had suffered from shortness of breath, pains in the chest and cold in the head. He had no venereal or genito-urinary infection but complained bitterly of the pain and burning on urination which he said was worse when he was on duty. He had given up his work for a few days but was offered a little better position and returned to the plant. His symptoms became so much worse that he entered the hospital. At the time of entrance to the hospital he was suffering from pain all over the chest and a severe cough. Physical examination showed the presence of moist râles throughout both lungs, most marked over the bases, and a prolongation of the expiratory note. He had little or no sputum. His temperature varied from 99° to 100° for the first three weeks and then became normal. The urine showed a slight trace of albumin, a positive Webster's test, a strong test for urobilinogen, no evidence of bile and microscopically a few hyalin and granular casts. The red blood count was 5,230,000; the white count was 10,000; the hemoglobin, 105 per cent.; and the differential count showed polymorphonuclear leucocytes 68 per cent., lymphocytes 18 per cent., and large mononuclear leucocytes 10 per cent. The first X-ray report suggested a cavity in the left apex with marked infiltration of the bronchi. The second X-ray plate made one week later showed marked thickening in the region of the hilus, with infiltration of the bronchi and mottling throughout both lungs. In another week there was no evidence of consolidation or suggestion of cavitation, but the prominence of the bronchial tree and mottling was still present. Two weeks later the chest was entirely negative. Recovery was apparently complete.

The striking things about the case were the slow onset of the irritative bronchitis without other clinical evidences of infection and the great prominence of the bronchial tree on the radiogram, with the gradual fading of these shadows as the patient's condition improved. In the light of the findings in Wagner's case it was suggested that healing took place by partial organization and retraction of the exudate in the bronchi.

Briefly stated, the lesions in acute gassings appear to consist in the injury to the surface epithelium, the exudation of serum, the invasion of organisms from the mouth and upper air passages, and the development of a more or less persistent bronchitis and bronchiolitis, with an occasional instance in which the bronchial walls are so severely damaged that hemorrhage and fibrinous exudation take place, the basement membrane is destroyed, and healing occurs by the organization of the exudate. The condition, while not due to T.N.T., must be considered with T.N.T. poisoning because it is a form of gas poisoning incidental to the manufacture of T.N.T. Steps to prevent it must come up for consideration when the means for controlling the dangers to the manufacturers of trinitrotoluene are thoroughly worked out.

T.N.T. Dermatitis. — T.N.T. is a local skin irritant and tends to produce staining, redness, hardening and scaliness of the skin. Instances of dermatitis have been described by White (15), Putnam and Herman (12) and others (16), (17). I personally saw several instances which were complicated by the ordinary chaffing due

to exposure of the hands in winter weather. One of these instances showed deep cracking and crevicing.

We attempted to produce T.N.T. poisoning in guinea pigs and rabbits by rubbing them with dry T.N.T., with wet T.N.T., and with T.N.T. suspended in oil. Dermatitis was developed more or less readily by each method. We purposely selected the back between the shoulders for the point of inunction because it was difficult for the animals to irritate the area by scratching. The skin became thickened, dry, scaly, the hair dropped out, and finally thick folds, or rugae, were formed. We also anointed several rabbits' ears because the loose tissues of the back were not very favorable for obtaining good sections. The ears passed through similar stages. When the skin was removed the subcutaneous tissues were always found to be stained yellow. Microscopically, the animal lesions resembled mild burns. The stratum corneum was thinned out and broken in places; the crusts were superficial and did not reach through the epidermis. The Malpighian layer was edematous, the fixed cells were swollen and the layer was infiltrated with a few polymorphonuclear leucocytes. Between the Malpighian layer and the papillae there were spaces filled with serum and polymorphonuclear leucocytes; the papillae and deep connective tissue fibers of the corium were edematous, the collagen fibrils were swollen, and the spaces between them were infiltrated with leucocytes, many of which were flattened out and appeared spindle shaped from pressure. A few plasma cells were present. The ducts of the sweat glands and hair follicles were profusely infiltrated with acute inflammatory cells.

Minor Poisonings and Severe Toxemias. — Many of the animals undoubtedly passed through phases known in the human as "minor poisoning" and "severe toxemia," but we were unable to differentiate these stages. Diarrhea was a very common early symptom in the animals fed T.N.T. and was particularly noticeable in those fed oatmeal crackers soaked in milk saturated with T.N.T. Later on, constipation was the rule.

Toxic Jaundice and Acute Yellow Atrophy of the Liver. — The most serious effect of absorbing T.N.T. is the toxic jaundice and acute yellow atrophy of the liver which has been found associated with it. Twenty-nine autopsies on cases of this kind have been reported; by Spilsbury (18) seven cases; Thursfield (2) one case; Livingstone-Learmonth (16) one case; Martland (4) one case; Stewart (19) eight cases; O'Donovan (3) two cases; Oertel (20) one case; Turnbull (21) three of his own and four submitted cases; and our own case (8). All of the clinical histories are more or less similar, and the characteristic findings may be summarized as follows. The employees recovered from the initial nervousness and early toxic symptoms common in beginners and continued to work for several months in comparative comfort before the severe toxic symptoms, accompanied by jaundice, appeared. A few of them had quit the T.N.T. factory and sought other work. As the jaundice developed, the affected individual experienced headache, general uneasiness and an itchiness of the skin. Medical examination revealed an elevated temperature, and a thorough urine examination gave positive tests for bile or bilirubin, a strongly positive Webster's test, and generally a positive test for albumin. Later the toxemia increased and vomiting, subcutaneous hemorrhages and pain over the liver developed. Hemorrhages from the lungs and stomach were less constant signs and, finally, localized edemas appeared, the liver dullness decreased greatly in size, and death ensued.

The striking thing at autopsy in these cases was the very small, flabby liver which showed masses of red necrosis with remain-

ing portions of yellow liver tissue greatly infiltrated with fat. The left lobes were constantly more extensively affected than the right, and the weights varied from about 400 to 1200 gm. The gross picture of all of the cases was characteristic of the classical acute yellow atrophy. Other important findings in nearly all of these cases were general icteric staining of the skin and of all the viscera, acute toxic nephritis, acute congestion of the spleen with enlargement, and petechial hemorrhages of pericardium, pleura and stomach mucosa. A few instances of hemorrhagic infarction of the lungs were reported. The bone marrow was found hyperplastic three times out of six examinations.

Histologically, the liver lesions are probably identical with those of so-called acute yellow atrophy. Several observers made early attempts to show that there was a difference in the amounts of fat and in the microchemical reactions of the fat present in the two conditions, but is it probable that the only differences were individual variations, which were dependent upon the stage of degeneration of the liver at the time of death and not upon any actual differences in the types of degeneration found.

Attempts to Reproduce the Liver Changes in Animals

Trinitrotoluene was administered to fifty-one animals, not counting controls, in the endeavor to produce acute yellow atrophy. Guinea pigs and rabbits were generally used, but one small series of dogs was tried. Nineteen out of twenty-eight guinea pigs exhibited liver lesions at autopsy but only one instance could be justly called acute yellow atrophy. We found rabbits and dogs less susceptible but we did not use Voegtlin's method in feeding the latter animals. The guinea pigs received relatively larger doses than the other animals.

T.N.T. was given to the animals by feeding in various ways, was injected subcutaneously in oil, was anointed in lanolin, and was dissolved in serum or normal salt and injected intravenously. Of all of these methods we had the best results in placing a small amount of powder on the back of the tongue by means of a glass tube and by following it with a drink of water or milk, and then holding the animal's nose until the material was swallowed. In one series, 10 per cent alcohol was used instead of water, because of the popular belief that the drinking of alcohol aggravates T.N.T. poisoning, but we obtained no evidence to the effect that poisoning progressed more rapidly or was more severe when alcohol was used. At first each dose was weighed carefully but this was given up as useless because the animals usually succeeded in spitting out a part of the powder and it was not possible to tell how much was actually swallowed.

In both guinea pigs and rabbits symptoms began to appear in from three to seven days. Guinea pigs showed earlier and more constant blood changes, while rabbits were found of value chiefly because of the ease with which they were catheterized and the urinary changes followed. Some animals died soon after symptoms appeared and some were kept in various stages of partial poisoning for weeks or months.

Symptoms of Experimental Poisoning. — The symptoms of experimental poisoning were fairly constant for both guinea pigs and rabbits, but were more exaggerated in the former. A description of the typical changes noted in guinea pigs covers the ground for both animals.

After the guinea pigs had been fed with T.N.T. for two or three days, brownish liquid stools or unformed fecal masses were found in the cages but after a few more days only scanty hard scybala were seen. Blood counts made in from four to eight

days showed a moderate anemia. T.N.T. was sometimes stopped at this stage and the animals always recovered. After resting for several days, the feedings were resumed, and so on. In one series of animals, T.N.T. was given daily for some time and these animals exhibited mild symptoms for several days and then suddenly became emaciated, lost from 100 to 200 gm., and showed evidences of marked blood destruction. When the animals in the latter series began to stay in one position and refused to eat, T.N.T. was discontinued for a time. Some of them recovered and others grew steadily worse and died in a day or two. Just before death they would sit shivering in one position and wobble from side to side as if drunk. The mucous membranes became purplish-yellow, the eye grounds blanched and the soles of the feet became deeply cyanotic. The heart action became slow and sluggish, and it was almost impossible to get blood enough from the ear to make a satisfactory count. The temperature was subnormal, 94° F. or less by rectum. Loss of bladder control and dragging of the hind legs were sometimes seen. One pig developed well-marked clonic convulsions for two hours before death.

Gross Autopsy Findings. — At autopsy, the following findings were commonly seen in guinea pigs. Anointed animals constantly showed a yellowish discoloration of the subcutaneous tissues beneath the area of inunction. The brain and central nervous system showed no gross changes. The heart was dilated, and the serum from the heart was yellowish-green in color. The lungs were yellowish and partially collapsed. The stomach was distended and so soft that the wall tore at the slightest manipulation. The intestines were generally empty. The liver was enlarged, pale yellow and fatty and contained sharply defined white foci with red borders. The gall bladder was, as a rule, well filled and the contents were often thin and opalescent. The spleen was enlarged, congested and black in color. The adrenals were orange-yellow. The kidneys were deeply bile stained. The skeletal muscles were generally yellow and bile stained. The bone marrow was deep purple in color and solid in consistency.

A condition similar to acute yellow atrophy of the liver was found in one guinea pig. This animal had been fed T.N.T., off and on, for nearly five months and, at one time, had recovered from a stage of poisoning which had proved fatal to all other animals. The liver, in general, was a bright yellowish butter color, divided up by intersecting red lines. Two of the lobes were shrunken and contained large reddish lesions with a puckering of the other liver tissue which radiated toward these reddened foci. The other organs resembled those found in all severe T.N.T. poisonings.

Rabbits presented similar but less marked symptoms and those which died from the effects of T.N.T. presented liver, spleen and kidney changes similar, both macroscopically and microscopically, to those found in the guinea pigs. Hyperplasia of the bone marrow in them was found many times.

Microscopic Changes in Experimental Lesions

Sections of the *brain*, *heart*, and *skeletal muscles* showed no convincing changes.

Lungs. — The lungs were in part collapsed and often edematous. One or two instances of early pneumonia were seen and some of the lobules were hemorrhagic. In the animals in which T.N.T. was forced into the lungs, large phagocytic endothelial cells were found in the alveoli and these contained both black granules due to anthracosis and coarse yellowish granules which did not give the iron reaction.

Liver. — The liver lesions were of several kinds, varying largely with the duration of

the poisoning, and may be separated, for purposes of description, into focal necroses, fatty changes and diffuse areas of necrosis.

The earliest lesions seen were small sharply defined foci of necrosis. These varied in size from the involvement of a few cells only, to about one-third of a lobule and were located near the central vein. Some of these small foci appeared to contain a few fibrin strands and were infiltrated with polynuclear and endothelial leucocytes. Some were simply sharply demarcated islands of cells which took eosin but no nuclear stains and were not infiltrated with inflammatory cells. A third type of focal necrosis consisted of a small area of cells which appeared to have undergone coagulation necrosis and chromatolysis. The cells of these areas stained irregularly and the nuclear material presented the appearance of flowing from one cell to the next. These lesions resembled the necrosis of eclampsia save that none of them was found near the periportal connective tissue.

FIG. 1. — Liver of guinea pig which had been poisoned repeatedly with T.N.T. for a period of nearly five months. Magnification is Zeiss AA Objective, No. 2 eyepiece. The left half of the figure shows a portion of the liver which has undergone extensive fatty infiltration, the interlobular connective tissue apparently being increased. The right half of the figure shows a portion of the liver in which the lobules are wholly necrotic, the stroma and cell spaces filled with blood alone remaining. Projecting into this area from the upper right-hand corner is an island of periportal connective tissue showing the proliferation of bile ducts. The gross appearance of the lobe from which this tissue came was that of acute yellow atrophy.

In the animals where the poisoning was carried out over a more prolonged period, the liver lobules showed advanced fatty

changes of both globular and granular types. The globules were found in the cells about the central vein and in some instances were seen to some extent throughout all of the lobule. In the very advanced fatty changes the inner one-half to one-third of the lobule was infiltrated with large fat globules and the cells were so greatly swollen that sinusoids appeared to have been compressed so tightly as to exclude the red blood cells from this portion of the sinus. Occasional large phagocytic endothelial cells filled with brown pigment remained to mark the positions of the sinusoids in such areas. Outside of this globular fatty area there were cells showing granular change, which Sudan III stains showed to be finely granular fat. The cells immediately about the periportal areas appeared to be infiltrated the least and the blood sinuses in these areas were usually congested. Associated with the fatty changes were fairly large areas of necrosis. These areas were found adjoining the central vein, in the mid-zonal regions and were common just beneath the liver capsule. The cells in these areas stood out bright red with eosin-methylene blue stains. In some places the cells seemed to have disappeared entirely, while the stroma which remained stained much redder than did the stroma of the more normal portions.

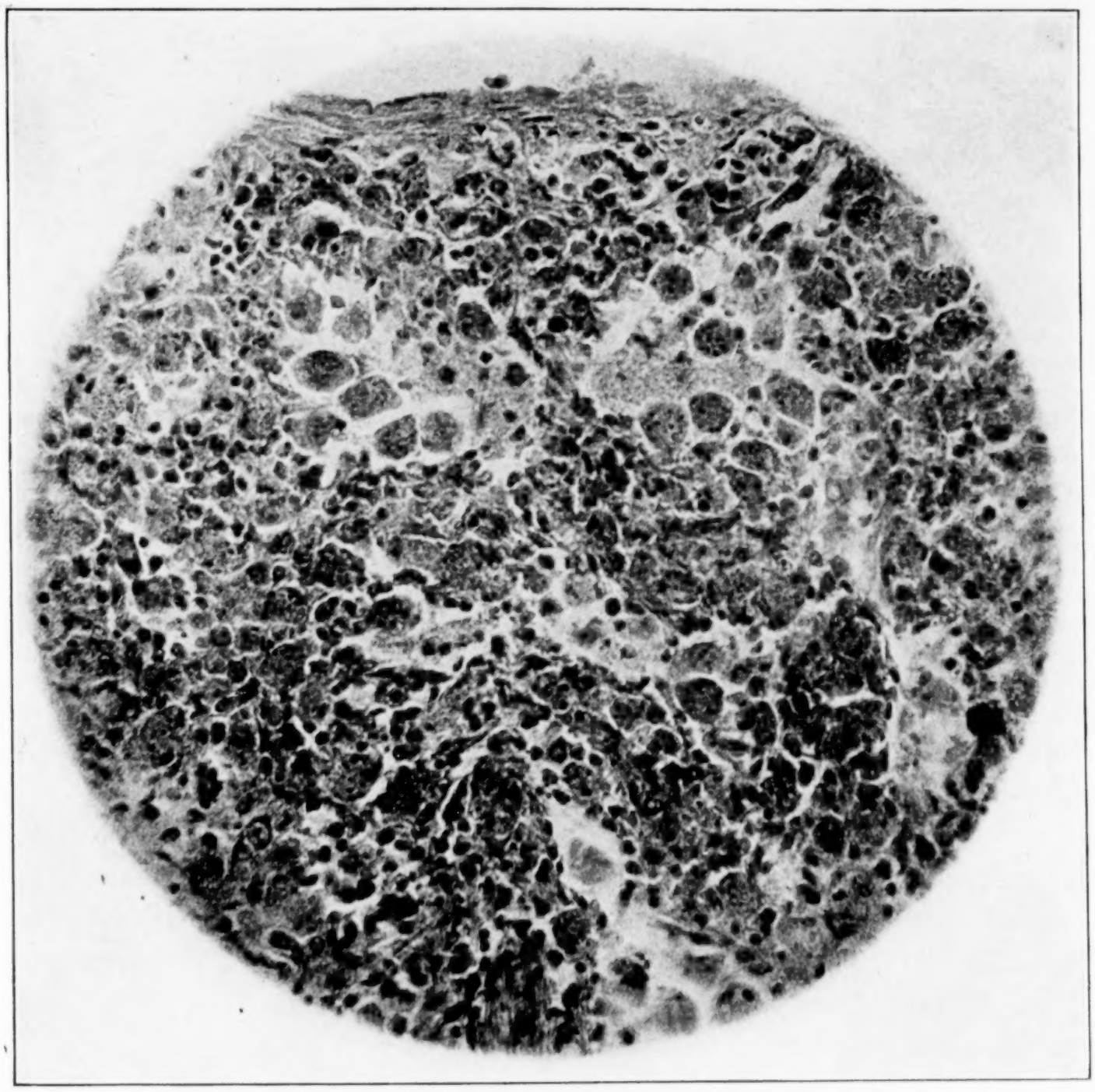

FIG. 2. — Section of the spleen of a guinea pig poisoned with T.N.T. Magnification, Zeiss DD Objective, No. 4 eyepiece. Note the large endothelial leucocytes, phagocytic for red cells, cellular debris, and blood pigment.

The third type of lesion was similar to focal necrosis, save that it was diffuse and

involved a number of lobules or at least the greater part of a lobule. Here the cells retained their normal arrangement and outlines but took only the diffuse stain. The borders of these areas were more or less infiltrated with inflammatory cells and were surrounded by areas of liver cells filled with fat globules.

In the liver which was grossly "yellow atrophy," all of the remaining liver cells found in the areas which had appeared yellow in the gross, were distended with fat globules. The red areas were made up of groups of necrotic lobules in which only the outlines of the stroma, the periportal connective tissue bundles, and the structures contained within them, were recognizable. In one animal, which had recovered from a very severe poisoning, the periportal bundles appeared to have approached much nearer to each other than normally. They contained a definitely increased amount of connective tissue together with numerous bile ducts. This finding suggested that repeated repair of the T.N.T. liver necroses would lead to cirrhosis.

Spleen. — The microscopic changes in the spleen of animals which were repeatedly poisoned and those which survived a considerable number of doses were constant and fairly characteristic. The blood sinuses became enlarged and quite definitely outlined. Most of the free red and white cells disappeared from the sinuses and their places were taken by large phagocytic endothelial leucocytes filled with golden pigment, blood fragments and whole red cells. These large cells arranged themselves in columns along the fine filamentous trabeculae and were also found free in the spaces. The appearance was suggestive in a way of Gaucher's spleen. The central portions of many sinuses surrounded by these large cells were clear open spaces. The only individual variations in the spleens which were free from postmortem changes were in the relative amounts of pigment and red corpuscles contained within the cells. In some phagocytes the pigment greatly predominated while in others the red cells were seen almost to the exclusion of the pigment. The pigment gave a strong positive iron reaction. While many of the spaces contained nothing but the large phagocytes, others contained a few red cells and an occasional normoblast. In a few instances normoblasts together with other red cells were seen incorporated within the large phagocytes. Nucleated red cells in the spleen were present in about the same proportion as in the vessels of other organs. The Malpighian follicles, vessels, and trabeculae appeared normal.

Adrenal Glands. — While most of the adrenals showed no microscopic changes there were several instances where small groups of medullary cells had entirely disappeared and the spaces formerly occupied by them were filled with free red cells.

Kidneys. — In all of the severely poisoned animals the histologic changes in the kidneys were similar. The glomeruli were congested and the glomerular spaces dilated and filled with cellular debris, free pigment granules and occasionally with the shadows of red blood cells. Some of the convoluted tubules were dilated and filled with red blood cells, red staining granular material and brown granules, and others had their lumina closed by the approximation of the swollen epithelial cells which, Sudan III stains show, contained granular fat. The straight tubules were dilated and filled with large casts which were made up chiefly of brown granular material, though they sometimes contained recognizable red blood cells. As a rule the interstitial tissues were not infiltrated with cells and the vessels appeared normal.

Bone Marrow. — The bone marrow was always taken from the hollow portion of the bony shaft of the femur. The amount

of fat present was constantly much less than that found in a series of specimens taken from supposedly normal animals, though there was a considerable variation in the amount seen in the normal series. Some specimens from T.N.T. animals showed no fat vacuoles while others showed a few which were widely separated. The megakaryocytes and multinucleated giant cells were numerous and often appeared arranged in groups of three or more. The latter type of cell was especially numerous, generally containing a great many large round vacuolated nuclei. These cells were not found to be phagocytic for either red or white cells. Many large groups of nucleated red forms were found and these cells appeared to occur in nests, though we were unable to determine that the normoblasts were peripherally located and the megaloblasts centrally situated, etc. These nests often contained all types of nucleated red forms. Some were large red cells with a greyish or purplish protoplasm and a large nucleus in which the deep blue chromatin strands were slightly separated. Some were equally large cells with clear red protoplasm and a deep blue compact nucleus. Some were typical normoblasts and some were normoblasts with two or three nuclei or with large lobulated nuclei, showing from three to five lobulations. An occasional microblast was found. Of the myelocyte series the coarsely granular eosinophilic myelocytes were the most common cells and they constantly occurred in nests or groups. A few polymorphonuclear eosinophilic cells were present among them. Other forms of myelocytes and leucocytes were abundant and a smaller number of lymphocytes were present. Occasionally large endothelial leucocytes filled with red blood corpuscles were seen, but they were not numerous in any specimen. One thing was definite about them and that was that they were able to phagocyte nucleated red forms as well as ordinary erythrocytes.

Chemical Tests

Numerous chemical examinations were made on the blood and urine of the animals during life and on the serum, bile, urine and tissues after death. Tissue extractions made in various ways were uniformly negative with Webster's test.

Chemical Examinations of the Blood. — The blood specimens for chemical examination were aspirated from the heart both before and after death. Just before death the blood became a deep red-brown or chocolate brown color and was rather thin. It was believed that the color was due in part to bile and in part to methemoglobin. Spectroscopic tests for methemoglobin in the blood were not convincing. The serum which was separated off from the blood was constantly stained a deep green bile color. While it was a constant finding that T.N.T. added to blood gave a positive Webster's test, we did not obtain an undoubted positive Webster's reaction on the blood or serum of a poisoned animal.

Bile. — Bile was aspirated with a clean syringe from the gall bladders of all of the animals which died of T.N.T. poisoning. The bile was sometimes thick and green and sometimes clear and almost opalescent. One undoubtedly positive Webster's test was obtained on bile and many doubtfully positive tests were reported.

Urine. — Urine specimens were obtained from the rabbits during life by catheterization, and from both guinea pigs and rabbits after death, by aspiration of the bladder with chemically clean syringes. These methods were used to exclude artificial contamination with T.N.T.

Appearance: The postmortem samples presented a fairly characteristic appearance. The urine was usually thick, opaque, and chocolate brown in color. Fresh specimens were acid. On standing, a heavy yellowish-brown flocculent precipitate sep-

arated out, leaving a distinctly bile stained reddish-brown fluid. Albumin was inconstant in catheterized specimens but was always positive in postmortem samples. Urobilin was often present in the antimortem samples and bile was positive in practically all of the postmortem samples. Webster's test usually became positive in two to three days in rabbits fed T.N.T., although there were instances in which the animal was definitely poisoned before the test was characteristic. Microscopically, the characteristic findings were great numbers of granular casts, some of which had red corpuscles adhering to them, and a large amount of brownish-yellow amorphous material, which cleared up on the addition of acetic acid.

Methemoglobinuria was suspected, because of the chocolate brown appearance of most of the specimens. We referred samples of urine from two guinea pigs and one rabbit to Dr. C. C. Guthrie of the University of Pittsburgh, for his opinion on this point. He reported that the urine of one of the pigs showed the presence of a distinct band in the red, occupying the position of methemoglobin, and of a diffuse band in the green, corresponding to reduced hemoglobin. The band in the red disappeared on the addition of ammonium sulphide. This sample had been centrifuged, but had not been otherwise treated. The second guinea pig urine which had been slightly acidified with acetic acid for purposes of clarification, gave the same spectroscopic reading, but the finding was a little less conclusive, because there was a possibility of some acid hematin having been formed by the addition of the acid. However, no excess of acid was used, the specimen was not heated, reduced hemoglobin was abundantly present, and the band in the red disappeared when ammonium sulphide was added. The rabbit's urine gave conspicuous oxyhemoglobin bands, a faint band in the red in the position of methemoglobin and no differentiation in the green to indicate reduced hemoglobin.

Dr. Guthrie concluded that the evidence was strong but not absolutely conclusive that methemoglobin was present in the urine of the two pigs, but that not more than a faint trace, if any, was present in the rabbit urine.

Excretion of T.N.T.

Based on the evidence furnished by Webster's test, the urine is undoubtedly the chief means of excretion of T.N.T. The statement of Voegtlin, Hooper and Johnson (11) that T.N.T. is excreted in the bile is borne out by our observations. We must, however, modify their statement that the feces never give a positive test. We obtained undoubted positive Webster's tests on the feces of all animals fed T.N.T. but did not get positive reactions in animals where T.N.T. was administered in other ways. This probably indicated that the bile is not an important vehicle of excretion from the quantitative standpoint.

Anemia

Anemia in T.N.T. workers is relatively common. Examples of mild toxic anemia, severe toxic anemia and aplastic anemia have been reported. Polycythemia (5 to 6 million cells) has been noted by Panton (22) and has been recorded by others in the very acute forms of toxic jaundice during the late stages.

Aplastic anemia was the first form reported, but subsequent studies seem to indicate that only a comparatively small number of cases are of this type. The blood picture was characterized by severe anemia with a high color index, by the absence of nucleated red cells and poikilocytes from the circulation and by an extreme leukopenia with a relative lymphocytosis. Purpura was a commonly associated condition.

Six autopsies on aplastic anemia due to T.N.T. have been reported. The bone marrow of these cases was either gray or fatty with areas of hemorrhage. The livers were generally not necrotic to the extent seen in toxic jaundice but showed extensive deposits of hemosiderin.

Toxic anemias of varying grades, ranging from mild changes in the appearance of the red cells and the decrease in the amount of hemoglobin to very severe anemia presenting a picture similar to pernicious anemia, have been reported by M. J. Stewart (19), L. H. Smith (23), Gregorson and Taylor (24), and by Minot (25). Minot's findings are very interesting in comparison with the experimental work and a fuller discussion of his results will be made later.

Experimental Anemia

The literature on experimental T.N.T. anemia is very meagre. Panton (7) reported an attempt to produce an aplastic anemia in guinea pigs by means of an injection of T.N.T. suspended in oil. He obtained negative results. Voegtlin *et al.* (11) described anemia as a constant finding in the dogs which they used in their work for the United States Public Health Service investigation. They summarized the blood findings in the animals as being a reduction in the total blood volume, a decrease in the red corpuscle volume and in the hemoglobin. They found an increase in the number of reticulated cells and in the number of nucleated forms, and mentioned the great inequality in the size of the erythrocytes.

In making routine blood counts on the animals in which we were studying the liver lesions it became apparent that the guinea pigs developed a severe anemia in almost every instance. We then decided to run additional series of both guinea pigs and rabbits for the special study of the blood changes. We knew from our work that the blood changes were fairly constant so that we felt that a small series of animals carefully followed was of more value than a large group which would be less tangible. In the rabbit series we used seven animals and several controls. Two were fed T.N.T., two were injected subcutaneously with T.N.T. in oil, two were injected intravenously with human serum saturated with T.N.T., and one was injected with salt solution saturated with T.N.T. After several doses the two animals which received serum died from anaphylaxis so that a new group of four rabbits was given the same treatment. These animals were given from four to nine intravenous doses, varying from 10 to 15 c.c. each, and none of them showed any blood destruction by blood count. The feeding animals developed a mild anemia, so that eventually all of the rabbits were fed T.N.T. and their bloods again studied. In rabbits the blood changes were inconstant and never severe. Usually the rabbits died from general toxemia before blood destruction advanced to any great degree.

The guinea pigs, on the other hand, showed early anemia which progressed to an extremely severe stage, and the changes were essentially the same for each animal. Ten guinea pigs and a control were used in this series. The animals were given T.N.T. by mouth until the blood changes appeared, and were then rested for several days, and the administration resumed. It was hoped by this procedure to exhaust the bone marrow and secure an aplastic form of anemia. At first the blood of several of the animals was counted on alternate days but later the counts were made only when the animals showed signs of toxemia and then the counts were made by heart puncture so that plenty of blood for fragility tests could be quickly obtained. The evidences of blood destruction became apparent in from three days to two weeks. When the anemia appeared early the animal was at

once rested and generally recovered, but when the symptoms were late the animal usually sickened rapidly and died, apparently from a cumulative effect. Two animals in which the blood counts were as low as two million recovered when rested.

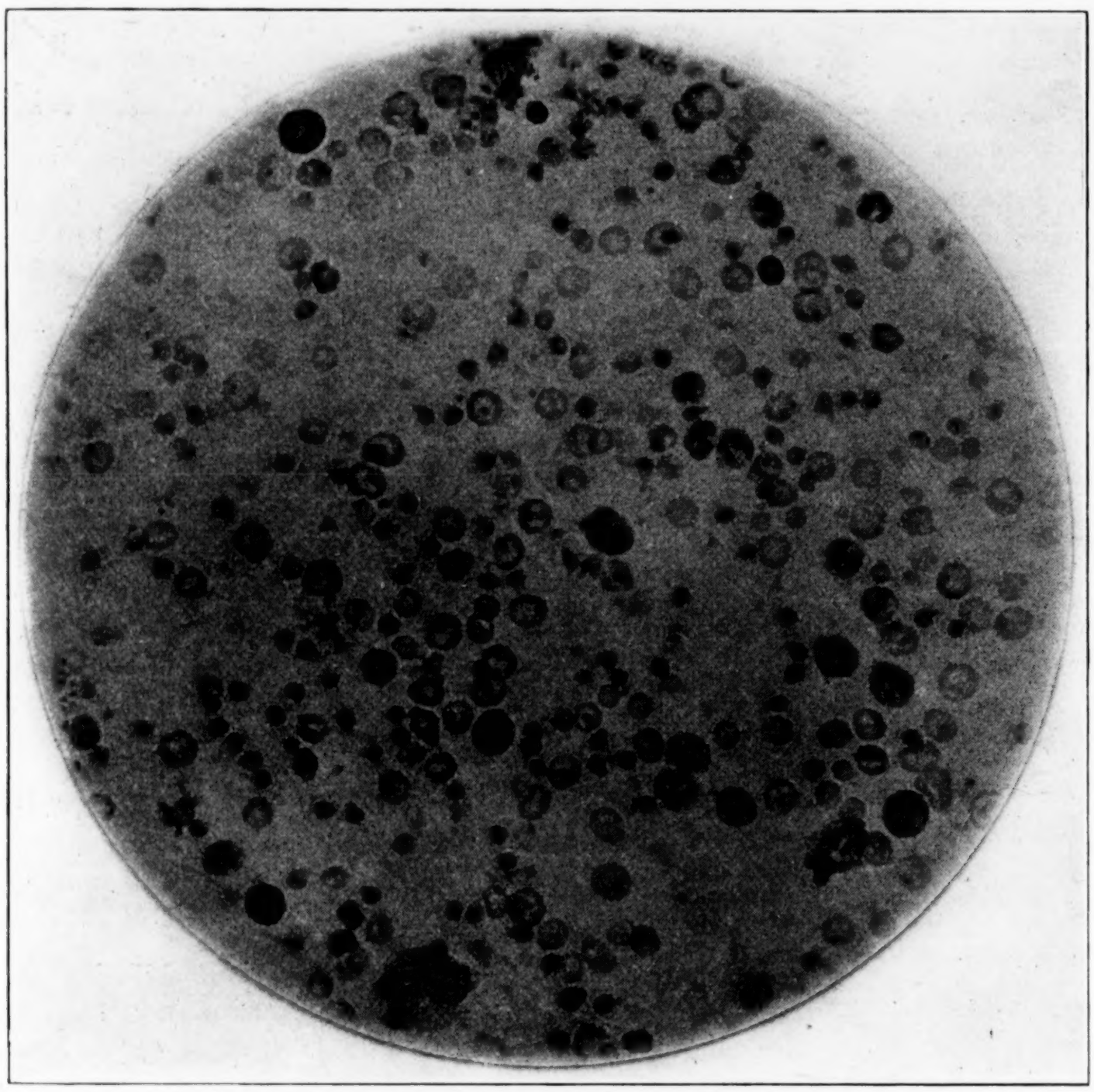

FIG. 3. — Photograph of blood smear from guinea pig poisoned with T.N.T., showing intense blood destruction. The field shows the great variation in the size of the red cells, numerous poikilocytes and several nucleated forms. Red cell fragments are also seen.

One of these two animals recovered rapidly. His count increased from 1,800,000 red cells to 3,600,000 in five days. The following synopsis indicates the changes observed in the blood:

Hemoglobin. — Estimations of hemoglobin were made with the Sahli hemoglobinometer. The old standard was used and the direct readings taken. The hemoglobin readings on unused animals ranged from 90 per cent. to 115 per cent. and on animals in the stage of poisoning from 35 to 50 per cent. One animal fell as low as 30 per cent. during the agonal period.

Red Cell Counts. — Counts on unused animals ranged from 4,500,000 to more than 5,500,000. The counts in poisoned animals were generally below 2,500,000. The lowest count was 1,200,000 cells.

Color Index. — The color index was greater than 1 in every instance after the number of cells fell below 2.5 millions. Indices ranging from 1.09 to 1.7 were found. The relatively high hemoglobin readings were probably due in part to the presence of abnormal hemoglobin combinations in the serum, as it is probable from the urinary findings that methemoglobin was present.

Abnormal Red Corpuscles. — During the severe toxic stage of poisoning, every guinea pig developed an anemia accompanied by the appearance in the circulation of all of the commonly recognized forms of abnormal red cells. Red-cell fragments were seen in practically every field in the advanced poisonings. Poikilocytes were numerous and made up a large percentage of the cells. Macrocytes were so numerous that practically every field contained several. Microcytes were present but were relatively uncommon.

Inequalities in staining of the red cells were very common. In some of the animals there appeared to be almost as many cells showing polychromatophilia as those which took the ordinary eosin stain. Anisochromia was much less marked and stippling was seen only in the cells of one animal, but in this instance it was well marked and unmistakable.

Nucleated red cells were very common, occasionally four or five to a field. In making the differential counts the number per hundred white cells was recorded. The highest number of normoblasts was 54, and of megaloblasts was 9 (see Table 1). Several different types of nucleated red cells were present; ordinary normoblasts were in the majority; large nucleated forms, much larger than normoblasts, came next; and only a few microblasts and Howell-Jolly bodies were seen. Some of the large nucleated forms were unusual in appearance, their cytoplasm being grayish-blue, like the darker of the polychromatophilic cells, and their nuclei being made of loosely woven coils of deep blue chromatin substances with orange-colored material appearing between the strands. Red cells with two and even three nuclei were found in the smears.

Reticulated Red Cells. — A drop of fresh blood was allowed to flow, or drop, into about an equal part of 1 per cent. brilliant cresyl blue solution, made up in physiological salt solution. After about two

TABLE 1. — BLOOD FINDINGS IN ANIMALS POISONED WITH T.N.T

Animal Number	Severe T.N.T. Poisonings								Recoveries		Second Poisonings	
	IA	I	II	III	IV	V	VI	VII	IV	VII	IV	VII
Per cent. hemoglobin (Sahli)	40	35	55	...	45	...	30	46	80	100	35	35
Red count in millions	1.7	1.6	2.7	...	1.8	...	1.2	1.4	3.6	4.6	1.5	2.0
White count in thousands	30	47	13	...	9	...	89	74	12	9	15	23
Polymorphonuclears	42	56	20	55	54	67	55	67	66	30	67	47
Lymphocytes	52	30	61	31	29	23	25	22	30	54	18	43
Eosinophiles	3	9	5	3	0	0	11	2	1	6	0	0
Basophiles	1.5	0	1	0.5	0	0	1	0.5	0	3	0	1
Large mononuclears (non-granular)	0.5	2	7	2.5	12	2	2	13	3	0	6	5
Large mononuclears (granular)	0	2	4	3	2	0	3	0	0	1	9	4
Myelocytes	0	0	0	0	3	8	3	4	0	0	0	0
Normoblasts per hundred	21	42	7	52	15	14	7	55	2	1	30	4
Megaloblasts	0	3	2	6	9	8	2	17	0	0	17	3
Multinucleated red cells	0	0	0	5	3	2	0	0	0	0	0	0
Poikilocytes	++	+++	+	++	++	++	++	++	0	0	++	++
Macrocytes	++	+++	+	+++	+++	+++	++	++	0	0	++	++
Microcytes	+	+	0	+	+	+	++	++	0	0	+	+
Polychromatophilia	++	+++	++	+++	+++	+++	++	++	0	0	++	++
Stippling	0	0	0	0	0	++	0	0	0	0	0	0
Per cent. reticulated cells	....	....	....	....	61	58	78	....	3?	2	50	17

Few = +. Moderate Number = ++. Many = +++.

minutes a small drop of the mixture was placed on a slide and immediately examined, and smears were made on cover slips for permanent mounts. The latter were allowed to dry in the air and were then counterstained with Wright's stain in the usual way and mounted in balsam. Counts were made on the permanent smears and the relative number of reticulated and non-reticulated cells determined. Unless a large number of red cells are counted the method does not always give accurate results, because there seems to be a tendency for the reticulated forms to occur in groups. Severely poisoned animals showed from 30 to 78 per cent. of reticulated cells. The increase occurred during the poisoning stage and the reticulated cells became less numerous as the animal recovered. When T.N.T. feeding was resumed the percentage of reticulated cells again increased.

White Blood Cells. — During the poisoning stage most of the animals showed an increase in white cells. One animal gave a count of 89,000 and another of 76,000, but the average was between 15,000 and 20,000 and some did not go above 10,000. Differential counts were of little value save as a guide to the number of nucleated red forms present. In the two high counts, polymorphonuclear leucocytes predominated; in practically all of the other instances there was a lymphocytosis up to 50 or 55 per cent. The counts are more fully shown in Table 1.

In two of the animals there were unidentified small ring-shaped bodies which stained with eosin and contained bits of bluish or greenish chromatin material.

Fragility Tests. — The red blood corpuscles of normal and poisoned guinea pigs were tested for comparative fragility in hypotonic salt solutions. The blood was withdrawn from the heart and washed once in 0.5 per cent. sodium citrate in physiological salt solution. The suspension was then brought up to the volume of blood originally withdrawn and 0.015 c.c. of the suspension added to each tube of hypotonic salt solution. The hypotonic solutions decreased by 0.025 per cent. from 0.800 per cent. to 0.200 per cent., and 1 c.c. was used in each tube. In a series of normal animals, the first traces of hemolysis began at about 0.575 and was complete either at 0.475 or 0.450. Similar tests were made on the blood of poisoned animals at a time when their symptoms were very marked and in each instance hemolysis appeared between 0.600 and 0.800 and was not complete even in 0.200, apparently indicating that the blood at this time contained cells both of greatly increased fragility and of greatly increased resistance. Two of the animals recovered from the acute condition and their resistance was then found to be increased so that the range was 0.450 to 0.350, instead of 0.575 to 0.450. When they were again fed T.N.T., their blood reacted as it had during the first poisoning — namely, beginning hemolysis between 0.700 per cent. and 0.800 per cent. and incomplete in 0.200 per cent. salt solution.

Effect of T.N.T. on Red Cells in Vitro. — Blood serum and physiological salt solution which have been saturated with T.N.T. do not hemolyze red blood cells *in vitro.* Sets of tritrations were run, using physiological salt and normal serum, saturated with T.N.T., in contact both with normal corpuscles and with corpuscles from poisoned animals; also, with serum from one poisoned animal to which T.N.T. was artificially added in contact with cells from normal and poisoned animals. No hemolysis occurred. Very faint traces of bile added to the titrations produced hemolysis, but bile is itself a hemolyzing agent.

Effect of T.N.T. in Blood Stream. — Repeated intravenous doses of serum and salt solution saturated with T.N.T. did not produce hemolysis in rabbits, nor any other

demonstrable effects upon the blood, or bone marrow.

Summary of Findings in Anemia. — The feeding of T.N.T. to guinea pigs produces a severe anemia not unlike pernicious anemia, which is apparently due to the destruction of blood in the circulation and which is unaccompanied by destructive lesions of the bone marrow. The hemolysis is not due to the direct effect of T.N.T. on the blood because T.N.T. does not cause hemolysis either *in vitro* or when injected directly into the circulation. The anemia has a 1+ color index, is characterized by the presence of all sorts of abnormal red cells in the circulation, and is generally accompanied by a lymphocytosis. The nucleated forms are present in the greatest numbers during the periods of feeding and almost disappear when the animal is rested. Reticulated cells also become much more numerous as the severity of the poisoning increases, and disappear as recovery takes place. Severe poisonings increase the resistance of the red cells to hypotonic salt solutions. All of the experiments produced a hyperplastic form of anemia without evidence of bone marrow exhaustion. Some animals were repeatedly poisoned for nearly five months and finally died of the poisoning without evidences of bone marrow aplasia. Prolonged poisoning with small doses was found impractical because of the cumulative effect of the drug.

Discussion of Findings in Anemia. — Generally speaking, the blood changes in the guinea pig experiments were in accord with those found by Minot (25) in his study of the blood of 233 trinitrotoluol workers. The findings in his group of thirty-three workers with severe blood changes were very similar to the changes in the animals during the severe toxic period. In both instances there was blood fragmentation, polychromatophilia, anisocytosis, lymphocytosis, the appearance of nucleated cells, and a great increase in reticulated forms. We were able to produce severe anemias and to poison repeatedly some of the animals, two of which were studied for five months, and yet the reaction was first and last a hyperplastic type of anemia. It is possible that the bone marrow could be exhausted in time and an aplastic form produced if we could graduate the doses accurately and eliminate cumulative action, but in our series there was no indication of aplasia. On the other hand, it is possible that other factors in addition to the T.N.T. poisoning may enter where aplastic forms occur — as for instance, a collateral infection of the blood stream, bone marrow, or some local focus of toxin production.

Discussion of Pathogenesis of T.N.T. Poisoning. — Trinitrotoluene is readily absorbed through the skin and along the alimentary tract. It may be excreted in the urine and to some extent in the bile as shown by the presence of Webster-positive substances. Though not a hemolytic agent when added to blood outside the body, continued absorption of it by the body leads to very evident blood destruction. This is shown by the decrease in the number of cells per cubic millimeter, the fall in the per cent. of hemoglobin, the presence of polychromatophilic cells, the phagocytosis of red blood cells by endothelial leucocytes (particularly noticeable in the spleen), the accumulation of hemosiderin in the liver and spleen, and by the appearance of bile in the urine. The mechanism of the initial hemolysis is uncertain, though it is probably associated with the formation of methemoglobin. Methemoglobin was not demonstrated in the early stages, but cyanosis was present, and the later finding of methemoglobinuria makes it the most likely cause of the interference with the oxygen-carrying power of the blood. It is probable that, in the animal work, excessive hemolysis once begun led to free hemoglobin in the blood stream, which, in turn,

acted to increase the secretion of bile (26). Later, the bile was found in abundance in the blood stream, and furnished another hemolyzing factor to augment the hemolysis. The bone marrow then became very active in order to compensate for the red cell destruction and many immature blood cells were thrown into the stream. If T.N.T. was removed at this time the bone marrow soon caught up, and recovery took place, but if T.N.T. absorption continued, the whole process became aggravated, visceral changes developed, and the condition advanced rapidly to a fatal termination.

Several factors may have united to bring about the development of the liver lesions. The liver necroses were so constant and so typical within certain limits, as to make it appear that T.N.T. exerted an almost specific influence on liver tissue. While it is possible that the lesions were due in part to the hyperactivity of the liver parenchyma, coupled with gradual asphyxiation due to the impaired oxygen-carrying powers of the blood, it is hard to rule out the direct action of T.N.T. because the bone marrow and kidneys were subjected to similar asphyxiation, were also forced to hyperactivity, and yet showed no comparable lesions.

There were no evidences of bone-marrow destruction in any of the animals, not even in those which had received intravenous injections. The nucleated red cells increased during the acute toxemic stages, and decreased during the resting or regenerating stages, just as Bunting (27) found in his animals after the intravenous injections of saponin, etc. He attributed the reaction to the destructive lesions of the marrow produced by the poisons, but it is obvious that his explanation is not applicable in our series.

The hyperplasia of the spleen was associated in some way with the excessive hemolysis. This is shown by the large amounts of blood pigment found in the spleen sections. The hyperplasia was accompanied by the appearance of large endothelial leucocytes filled with phagocyted red blood cells, nucleated red cells, fragments of red cells and blood pigment. We think it more likely that these large cells played a passive part, by phagocyting red cells, etc., already injured, than that they actively destroyed them.

The kidney lesions appeared to be of a toxic nature and probably were due, in part, to the excretion of toxic T.N.T. products and, in part, to the effect of the general asphyxiation on the renal tissue. During the later stages, hemoglobin, methemoglobin, and bile were excreted in addition to the Webster-positive substances. The final interference with kidney function played an important part in the fatal termination of the poisoning.

No hemorrhages, such as were seen in the clinical cases, were found in the animals.

Summary. — The chief factors leading to the terminal pathologic findings appeared to be hemolysis, probably associated with the formation of methemoglobin, hypersecretion of bile, general asphyxiation of all tissues, direct toxic action of T.N.T. on the liver, bone marrow hyperplasia, and final failure of kidney function.

Conclusions

1. Trinitrotoluene fed to rabbits and guinea pigs or rubbed into their skins, produces liver lesions similar to those found in human cases of T.N.T. toxic jaundice.

2. Methemoglobin appears in the urine of some animals experimentally poisoned with T.N.T., during the late toxic stages.

3. T.N.T. fed to guinea pigs produces a very severe anemia. The anemia is hyperplastic in type, has a +1 color index and is accompanied by the appearance in the circulation of all types of nucleated red cells and by an increase in the number of reticulated cells.

4. Experimental anemia in the guinea

pig, due to T.N.T. administration, presents the picture of a so-called primary anemia, but appears to be due entirely to blood destruction in the circulation, since repeated histologic examinations of the bone marrow failed to reveal any destructive lesions of the blood-forming centers.

5. The resistance of the red corpuscles to hypotonic salt solutions was found to be increased after the animals had recovered from severe poisonings.

6. Removal of the animals from exposure to T.N.T. usually led to complete recovery, even though a high degree of anemia had developed.

7. The similarity of the experimental findings to the blood changes in employees described by Minot, emphasizes the importance of thorough blood studies in instances of suspected T.N.T. poisoning. The hopeful prognosis in animals brings out the importance of early diagnosis and immediate removal of anemic persons from contact with T.N.T.

In closing the author wishes to express his thanks to Dr. T. McC. Mabon, who made many of the routine blood examinations, and to Dr. S. S. Waddell for her assistance in carrying out a number of the chemical tests.

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BOOK REVIEWS

Lectures on Industrial Psychology. By Bernard Muscio, M.A. (Sydney); M.A. (Gonville and Caius College, Cambridge); Late University Demonstrator in Experimental Psychology, Cambridge; Sometime Acting Lecturer in Mental and Moral Philosophy, Sydney. Cloth. Pp. 300 with illustrations and index. New York: E. P. Dutton and Company, 1920; London: George Routledge and Sons, Ltd., 1920.

This book, divided into five lectures, is well written and contains a wealth of interesting illustrations from actual field work in psychological problems related to industry.

Muscio defines psychology as "accurate and systematic knowledge about the mind" and proceeds to show that, although the application of psychology to industry is still very limited, there is a considerable body of fairly precise psychological knowledge which is relevant to industry. In medicine, education, and law such knowledge has been used extensively for some time. The reason for applying psychology to industry is to further the aim of industry, which is to produce the material goods required by civilized man in the most economical manner possible, by (a) selecting workers on the basis of natural fitness and (b) constructing good methods of work for the purpose of obtaining from any expenditure of human energy or effort a maximum production. From the standpoint of the employer, the maximum production is to be obtained from a fixed amount of human energy. From the standpoint of the employee, the quantity of production is more the fixed quantity, and is to be obtained from the minimum expenditure of human energy. The suggestions offered by industrial psychology can be carried out by one who regards the question from either point of view.

It is clearly stated that there is no similarity whatever between industrial psychology, as above defined, and speeding up, which is "the attempt, by offering incentives of one kind or another to the will, to induce operatives to expend more than the greatest reasonable amount of energy in a given time."

The second lecture takes up mental factors relevant to industry. First, the nature of fatigue is discussed from its physiological and psychological aspects, and some present industrial causes of fatigue are considered, with the effects of fatigue upon industry. For example, the influence of fatigue upon the quantity and quality of output is an important consideration, and many figures are shown and analyzed in connection with the shortening of working hours, industrial accidents, and production. Suggestions for decreasing industrial fatigue are given, for "the elimination of industrial fatigue will render possible for the industrial worker a mental and bodily condition which, though not constitutive of, is nevertheless prerequisite for, an ideal social life."

Not only by shortening hours is fatigue to be reduced but by better muscle co-ordination and by constructing good methods of work, e. g., so that as much use as possible shall be made of similar groups of muscles exercised simultaneously on the two sides of the body.

Lecture III discusses the selection of workers on the basis of natural fitness. Psychological work in this field has been done since 1909, although Taylor applied the principles forty years ago. Two main principles of selection are in use: (1) the *each-capacity principle*, in which the applicant is given a number of tests to find out his capacities in various lines thought to be relevant to the work applied for; and (2) the *central-capacity principle*, where one test is constructed which elicits the ability of the individual to perform in the laboratory a complex task psychologically similar to the work. The practicability of the theory of selection is clearly shown, although it is recognized that adult workmen trained in work for which they were never naturally fitted would have a legitimate objection to being tested and forced to change their vocation. On the other hand, there are no difficulties, except conservatism, in the way of applying the principle of selection to boys and girls who are about to become wage earners.

In the fourth lecture scientific management, "Taylorism," and industrial psychology are compared. When scientific management is at its best, its three main tasks are: (1) to determine scientifically the best possible conditions of work; (2) to induce workmen to accept the conditions of work that have been proved scientifically to be best; and (3) to teach workmen the new work methods devised as part of the best possible work conditions. Thus, the aim of scientific management is to obtain the greatest possible adjustment, and in this attempt industrial psychology becomes relevant to it.

Cases are given to show how new methods of work have decreased the labor and increased output; for example, the introducing of pauses into monotonous and tedious work, the substituting of automatisms for frequent acts of decision, and the modification of the actual movements of the workmen. Thus, industrial psychology has its place in scientific management.

The last lecture is on the desirability of applying psychology to industry, the chief points being as follows:

"We have been concerned with the question whether the application of psychology to industry is desirable. In considering this question, it was found advisable to discuss scientific management similarly, since it is only in connection with scientific management that psychology has been, or is likely to be, applied to industry. From a cursory examination, it seemed that the only persons who might find the application of psychology to industry undesirable were industrial workers. The answer to our main question thus required an analysis of the whole situation from labour's point of view.

"In the course of this analysis, we discovered that, without exception, all labour's important objections to industrial psychology were really directed against the method by which it had been put into practice. Certain of labour's objections, it is true, seemed to rest upon confused thinking; but there were two — the autocratic tendency of scientific management, and its relation to collective bargaining — which seemed based upon undoubted fact. It seemed, also, that labour's hostility, so far as it was based upon these two objections, was not unreasonable. If the matter had ended there, we should have been forced to the conclusion that, for labour, at least, the application of psychology to industry is undesirable.

"This, indeed, is where the matter rests at the present moment. Looking ahead, however, we see that certain modifications in the method by which the application of psychology to industry is made, are possible. These modifications consist in a further democratisation of workshops, and in offering to labour, perhaps in the form of a shorter working day, a larger share of the new profits than has hitherto been given it.

"If these modifications were introduced, the application of psychology to industry would probably seem to labour highly desirable, and the worker would be as eager as anyone to adopt the new discoveries. For the aim of labour is to increase its share of common human goods. Such an increase, of course, is not secured by simply increasing the productivity of the human energy expended in industry; but while this is obvious, it is equally obvious that the greater the profits the greater the possible share of them for labour. . . . If, then, labour's protective rules and institutions were safeguarded and if an unambiguous and considerable share of the new profits were accorded it, there seems no doubt that it would find the application of psychology to industry very desirable. . . .

"I shall conclude by emphasising the great fact about which these lectures have centred. This is that, by certain applications of psychology to industry, it has been found possible to obtain a given output from a much smaller expenditure of human energy than that hitherto necessary. This, I think, is a great fact: it is for men to say how they will use it. Psychology has now to turn to its laboratories again. Its task is to better what has been done. To devise practical methods by which its results, and those of other sciences, may be available for the enjoyment of all, is the task of society in general, and especially of labour and capital." — *Stanley Cobb.*